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AN APPRAISAL OF FOUR YEARS' EXPERIENCE WITH ROUND WINDOW GRAFTS FOR DEAFNESS

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In June 1937 a small series of cases was reported [1] in which the round window niche had been blocked with a tissue graft for alleviation of deafness. Later (May 1938) a second report [2] was made, detailing results in a slightly more extended group of cases, the oldest of which had been followed for twenty months after operation.

In the second report, presented at a symposium on the treatment of chronic progressive deafness before the American Otological Society, the discussion drew attention to the fact that normal variation in repeated audiograms might well account for the reported improvement in these operative cases. A subsequent study of this variation [3] and establishment of minimum standards of improvement have shown that the reported benefit was real and not simply a matter of applied statistical data.

This paper presents an analysis of still a relatively small series of operations, 36 in all. The cases included were observed and studied over approximately four years, the oldest case for three years and nine months and the most recent for six months. The present purpose is to correlate the preoperative diagnostic study with all the clinical factors involved, the established loss of hearing by both air conduction and bone conduction and the patient's subsequent course.

Obviously it is impossible to recite in detail the complete history of each individual case. However, salient factors relative to selection of operable cases will be mentioned, and statistical data for all patients and

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From the Otological Research Laboratory of the Abington Memorial Hospital.

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1. Hughson, W.: Grafts in the Round Window in the Treatment of Certain Types of Deafness, Arch. Otolaryng. **25**:623 (June) 1937.

2. Hughson, W.: Rationale, Technique, Case Reports and Observations with Grafts in the Round Window, Laryngoscope **48**:533 (Aug.) 1938.

3. Witting, E. G., and Hughson, W.: Inherent Accuracy of a Series of Repeated Clinical Audiograms, Laryngoscope **50**:259 (March) 1940.

the group now regarded as favorable in the light of this four years' experience will be considered.

As has been stated,[4] no audiometric improvement of the threshold of hearing representing less than a sustained level of 10 decibels can be regarded as of any real consequence. The range of improvement must be extended both below and above the generally accepted critical frequency range 512 to 2048 cycles. Immediate improvement in hearing following any operative procedure is probably of no ultimate significance. Twenty per cent of the patients whose cases are reported here had a substantial gain in their hearing level during the operative period, or the time in which the drum membrane remained open, while 80 per cent had impairment. With closure of the defect the hearing returned to its preoperative level.

This small series of cases is presented as a complete group and in groups differentiated from the standpoint of hearing loss, age and duration of deafness in relation to observed pathologic conditions. If it is possible to demonstrate a useful improvement in hearing after assignment to any one or all of these classifications, the final appraisal of operative benefit must be left to the individual otologist and to his patient.

One point must be given further reemphasis. The results of this particular operation are not dramatic and are not immediately apparent. No serious psychologic depression resulting from this enforced period of waiting has been observed. On the contrary, the seriously deafened person who has gone through the long routine of standard treatments (and very few have been operated on who have not had such a therapeutic prelude) is not unduly sensitive to a relatively short delay in the achievement of true subjective improvement in hearing.

The following data, derived from a sustained and continued study of operative cases, are presented largely on a statistical basis. In 6 cases operation was performed on both ears. The first operation was always done on the poorer ear. If as a result of operation this ear showed definite improvement after an interval of one year, the second operation was performed.

The original standards of operability included so-called normal bone conduction. It has been shown experimentally [5] and more recently by Crowe and Baylor [6] in clinical cases that bone conduction is not necessarily a measure of intact nerve function. The loudness balance test

4. Hughson, W., and Witting, E. G.: Estimation of Improvement in Hearing Following Therapy of Deafness, Ann. Otol., Rhin. & Laryng. **49**:368 (June) 1940.

5. Hughson, W.; Thompson, E., and Witting, E. G.: An Experimental Study of Bone Conduction, Ann. Otol., Rhin. & Laryng. **45**:844 (Sept.) 1936.

6. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. **112**:585 (Feb. 18) 1939.

developed by Steinberg and Gardner [7] and by Fowler [8] is an important adjunct to otologists' methods for determining true neural impairment. If recoupment is an accepted fact, which it must be, the rationale of the round window graft operation should make this procedure effective if any part of the neural mechanism of hearing still functions.

A compilation of all clinical facts and findings, together with averages of three preoperative audiograms and of the last three audiograms made in each case, follows.

In summarizing these data the following facts become apparent:

SEX AND DECADE

Twenty-one women and 9 men were operated on. The number of patients in each decade of life was as follows: second, 3; third, 5; fourth, 11; fifth, 10; sixth, none, and seventh, 1. Originally a maximum age of 50 had been set as the limit, and in this review the operative results more than confirm this standard. Indeed, if any change were made the age limit should be lowered, as the patients in the fourth decade showed improvement with greater consistency than the others, although in the second decade, represented only by 3 patients and 4 ears, improvement occurred in every instance. The possibility of operating on children in the first decade has been considered, but none has so far been operated on.

DURATION OF DEAFNESS AND PREVIOUS TREATMENT

The duration of loss of hearing is considered a matter of great importance. Obviously the probability of an increase in this factor would be related directly, first, to the patient's age and, second, to the type of pathologic process found. In this series loss of hearing had been apparent to the patient for an average of twelve and a half years. The shortest duration of deafness resulting from acoustic trauma was three months; the longest, thirty-six years (the patient had been deaf since infancy and had congenital absence of the stapes). Two other patients had been deaf for more than thirty years; 4 for twenty years or more; 6 for between ten and twenty years, and 14 from three months to ten years. In this group 23 patients had received previous treatment, in every instance dilations, inflations and bougienage and in a few various electrical and manipulative procedures. This average duration of twelve and a half years, with treatment in 80 per cent of the cases resulting in neither improvement nor arrest of progress, is an important fact. The 6 patients with no treatment had impairment of

7. Steinberg, J. C., and Gardner, M. B.: Dependence of Hearing Impairment on Sound Intensity, J. Acoust. Soc. America **9**:11 (July) 1937.

8. Fowler, E. P.: The Use of Threshold and Louder Sounds in Clinical Diagnosis and the Prescribing of Hearing Aids, Laryngoscope **48**:527 (Aug.) 1938.

hearing for three and a quarter years only. Again, from a purely chronologic standpoint, the younger the patient the shorter must be the duration of the deafness, and the younger group exhibits the most favorable operative results.

LOSS OF HEARING

The statement made in the previous reports that a loss of hearing greater than 50 decibles in the speech frequency range would render any possible improvement of unlikely benefit still holds, but here again if any modification is to be made it must be a decrease rather than an increase in this level. The average decibel hearing loss in 30 operative cases (36 ears) was:

Frequency.....	128	256	512	1024	2048	4096	8192
Average.....	37.8	41.2	45.9	50.9	47.8	53.6	51.2
Greatest impairment.	65	80	80	80	70	80	—
Least impairment....	10	25	25	25	25	10	15

Nine patients in the series who have shown no improvement at any time had hearing losses below the prescribed 50 decibel level in addition to such detrimental factors as age, congenital abnormality, postoperative tinnitus and excessive use of drugs (in 1 case, acetylsalicylic acid).

PATHOLOGIC CHANGE OBSERVED

In no case could the appearance of the tympanic membrane be considered normal except that of the patient proved at operation to have congenital absence of the stapes. Thirty-one ears showed either thickening or retraction of the tympanic membrane, or both, in varying degrees. Fourteen showed sclerosis of the posterior margin, and in 17 the light reflex was either absent or definitely restricted.

When the patients were first seen, tonsillectomy and adenoidectomy had been done in all but 2. In 1 of these the tonsils were removed before operation; the other showed no disease. In no instance was sinus disease a factor. Nine patients showed moderate hypertrophy of the middle turbinate or deviation of the septum but not enough to be considered a contributing factor in the loss of hearing. In 4 only was the eustachian orifice definitely restricted in size.

Various constitutional defects appeared in 12 cases: toxic thyroid, in 1 (under therapeutic control); hypothyroid conditions, in 5; definite allergy, in 3; adrenothyreopituitary deficiency, in 2; arteriosclerosis, in 1, and trifacial neuralgia with excessive use of acetylsalicylic acid, in 1. In 1 case, in which operation was performed before the report had been received, the Wassermann reaction was subsequently found to be positive. This did not complicate wound healing or retard recovery. In all other cases the Wassermann reaction was negative.

The hemoglobin level was relatively normal in all cases. Except in frankly arteriosclerotic hypertensive persons, deafness is associated with low normal levels of blood pressure.

Treatment other than surgical was employed only in the groups with endocrine and allergic diseases.

OPERATION

The operation as previously described [9] was carried out in 30 cases, with a total of 36 ears. No infection occurred, and the drum healed in every case even after actual resection of portions of the flap. Tinnitus, which was present in 15 cases, was partially relieved in 5. It was never completely cured. The single postoperative complication was the development of pulsation tinnitus in the patient with a toxic thyroid. The graft in this instance became vascularized very rapidly. When it was touched with a probe during the operative period there was troublesome oozing of blood for several minutes. In 5 cases, about one year after operation there appeared a slight area of atrophy in the tympanic membrane at the site of the operative incision. The appearance of this atrophic area was not associated with any change in the hearing level. As has been stated, the second operation was performed only after an interval of one year and then only if the hearing level of the operatively treated ear had become equal to or better than that of the other. Obviously a second operation must be a matter of the patient's own election and implies a genuine subjective sense of improvement.

Does the useful improvement observed warrant the operation, in consideration of the possible hazards? The answer to this question is direct and unqualified. In the light of this four years' experience the operation is essentially without hazard, at least so far as any surgical procedure may be so considered. The major risk is, of course, infection. No infection occurred in this small series or in any other type of procedure, carried out for whatever purpose, in either the external canal or the middle ear. The aseptic technic employed is simple and direct, involving only the standard routine of general aseptic surgery. The lack of postoperative infection in this series compares favorably with its incidence in any field of aseptic surgery. It is felt, therefore, that the surgical technic as already described does not present any hazard and that, from this point of view at least, there can be no contraindication to the operation.

Except in the case in which tinnitus developed postoperatively, no late complications have occurred. It is interesting to speculate on the fate of the round window membrane in the cases which have been

9. Hughson (footnotes 1 and 2).

TABLE 1.—*General Summary of Data*

Case, Age, Sex, Ear	First Seen / Operated On / Last Seen	Duration, Years	Cause	Family History of Deafness	Previous Treatment	Average of Three Preoperative Audiograms / Average of Three Final Audiograms: 128	256	512	1024	2048	4096	8192	Bone Conduction	Tinnitus	Examination: Otoscopic	Nose and Throat	Sinuses	General	Diagnosis
1 16 F R	7/29/36 8/26/36 1/25/40	12	C. C. O. M.	Negative	Inflation; bougienage	53 43	53 45	60 45	63 55	55 50	50 50	45 47	Impaired; = AC	Absent	T+ R+ S LC--	Normal; T & A	Normal	B. P. 110/70; Wass. neg.; underweight	Chronic catarrhal deafness
1 16 F L	7/29/36 7/13/37 1/25/40	12	C. C. O. M.	Negative	Inflation; bougienage	24 35	28 43	40 52	45 53	42 52	43 53	38 47	Impaired; = AC	Absent	T+ R+ S LC—	Normal; T & A	Normal	B. P. 110/70; Wass. neg.; underweight	Chronic catarrhal deafness
3 45 F L	10/27/36 1/ 7/37 10/12/38	34	Colds	Negative	Dilation, inflation; bougienage	38 37	47 45	43 45	47 55	50 50	57 50	70 47	Normal; > AC	Present	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 118/90; Wass. neg.; neuritis N. acetylsalicylic acid Vth : acetylsalicylic acid	Chronic catarrhal deafness
4 21 F L	8/26/36 7/15/37 1/22/40	7	Postoperative (T&A)	Sister 2 aunts	Dilation; inflation; osteopathic treatment	28 35	38 40	43 47	37 43	45 42	65 67	75 75	Normal; > AC	Present	T+ R+ S LC+	Normal; T & A	Normal	B. P. 114/70; Wass. neg.; overweight	Chronic catarrhal deafness
4 21 F R	8/26/36 9/16/36 1/22/40	7	Postoperative (T&A)	Sister 2 aunts	Dilation; inflation; osteopathic treatment	35 27	45 37	53 42	43 40	62 47	63 65	75 70	Normal; > AC	Present	T+ R+ S LC+	Normal; T & A	Normal	B. P. 114/70; Wass. neg.; overweight	Chronic catarrhal deafness
6 26 F R	1/20/37 4/ 1/37 4/17/40	6	Colds	Grandmother; great-aunt	Dilation; inflation; bougienage	53 50	62 60	63 58	60 65	43 42	60 43	40 30	Normal; > AC	Present	T+ R++ S LC—	Normal; T & A	Normal	B. P. 128/85; Wass. neg.; B. M. R. —10; Hgb. 75%	Chronic catarrhal deafness
7 24 F R	3/28/36 2/ 2/37 4/ 3/40	6	C. C. O. M.	Negative	None	37 37	47 38	48 47	50 52	48 57	28 33	32 38	Normal; > AC	Absent	T+ R+ S LC—	Normal; T & A	Normal	B. P. 118/80; Wass. neg.;	Chronic catarrhal deafness
8 29 M R	11/ 7/36 2/18/37 3/16/40	2	None	Brother; 2 sisters	Dilation; inflation; bougienage	36 28	42 38	48 47	42 52	48 57	75 33	75 38	Normal; = AC	Severe	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 122/76; Wass. neg.; B. M. R. —15; hypothyroid	Chronic catarrhal deafness
9 35 F R	6/10/36 10/ 7/36 1/24/40	30	Familial	Brother; 2 sisters; brother; grandmother	Dilation; inflation; bougienage	67 63	75 70	78 78	70 75	63 65	50 50	57 57	Impaired; = AC	Present	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 108/64; Wass. neg.	Hereditary deafness

35 F R	10/ 7/36 1/24/40			2 sisters; brother; grandmother	inflation; bougienage	61 63	75 70	78 78	70 75	63 65	50 50	57 57	Impaired; = AC	Present	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 108/64; Wass. neg.	*Hereditary deafness*

10 39 F R	3/ 9/37 4/13/37 11/28/39	18	C. C O. M.	Negative	Dilation; inflation; bougienage	47 45	50 53	58 52	62 65	50 57	47 55	33 42	Impaired; = AC	Absent	T+ R+ S LC—	Normal; T & A	Normal	B. P. 108/70; Wass. neg.; hypothyroid	Chronic catarrhal deafness
11 39 F R	11/ 4/37 1/13/38 2/ 7/40	10	C. C. O. M.; colds	Negative	Dilation; inflation; bougienage	35 38	38 48	43 38	50 55	43 43	65 55	32 28	Normal; > AC	Severe	T R+ S LC+	Normal; T & A	Normal	B. P. 120/70; Wass. neg.; allergic	Chronic catarrhal deafness
11 39 F L	11/ 4/37 7/25/39 2/ 7/40	10	C. C. O. M.; colds	Negative	Dilation; inflation; bougienage	38 47	42 50	48 57	53 67	40 47	68 68	42 45	Normal; > AC	Severe	T R+ S LC+	Normal; T & A	Normal	B. P. 120/70; Wass. neg.; allergic	Chronic catarrhal deafness
12 44 F R	8/ 8/37 9/20/38 3/28/40	38	C. C. O. M.; colds	Negative	Dilation; inflation; bougienage; vaccine	23 25	27 28	23 22	27 28	47 43	70 77	67 70	Impaired; = AC	Present	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 112/80; Wass. neg.; B. M. R. —5; Hgb. 80%	Chronic catarrhal deafness
12 44 F L	8/ 8/37 9/21/37 3/28/40	38	C. C. O. M.; colds	Negative	Dilation; inflation; bougienage; vaccine	28 27	37 35	27 27	40 45	41 43	68 67	75 70	Impaired; = AC	Present	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 112/80; Wass. neg.; B. M. R. —5; Hgb. 80%	Chronic catarrhal deafness
14 47 F L	6/22/37 11/30/37 7/19/39	27	C. C. O. M.; colds	Sister	Dilation; inflation; bougienage	33 28	40 35	43 38	43 48	62 48	77 72	75 75	Impaired; = AC	Present	T+ R+ S LC—	Hypertrophied turbinate	Normal to roentgen examination	B. P. 122/85; Wass. neg.	Chronic catarrhal deafness
15 40 F R	10/ 2/37 10/21/37 6/ 9/39	7	Colds	Mother; sister	None	33 25	27 23	37 27	48 37	32 25	33 28	38 23	Impaired; = AC	Present	T+ R+ S LC—	Normal; T & A; pharynx congested	Normal	B. P. 112/74; P. 68; B. M. R. —4; Wass. neg.; hyperostos's of skull and sella	Chronic catarrhal deafness
16 41 M L	1/18/38 2/15/38 4/30/40	8	A. O. M.	Negative	Dilation; inflation; bougienage	52 48	55 53	57 53	50 50	43 58	42 52	42 53	Impaired; = AC	Present	T+ R+ S LC—	Normal; T & A	Normal	B. P. 115/70; P. 64; Wass. neg.; hypothyroid	Chronic catarrhal deafness
17 16 M R	12/ 7/37 3/10/38 9/25/39	3	C. C. O. M.	Negative	None	38 35	40 40	48 42	42 43	43 45	52 42	48 32	Normal; = AC	Absent	T+ R+ S LC—	Normal; T & A	Normal	Wass. ++; died 12/24/39 of pneumonia	Chronic catarrhal deafness
18 35 M R	4/13/37 4/ 9/38 12/13/39	10	None	Negative	Dilation; inflation; bougienage; thyroid	50 47	57 53	55 48	58 62	55 40	63 62	38 23	Normal; > AC	Absent	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 114/70; P. 56; B. M. R. —20; Wass. neg.; hypothyroid	Chronic catarrhal deafness

The following abbreviations are used: A.O.M., acute otitis media; C.C.O.M., chronic catarrhal otitis media; AC, air conduction; B.P., blood pressure; Wass., Wassermann reaction; T&A, tonsillectomy and adenoidectomy; B.M.R., basal metabolic rate; P., pulse rate; Hgb., hemoglobin; T, thickening; R, retraction; S, sclerosis, and LC, light reflex. The plus sign indicates the presence of the condition referred to; the minus sign, its absence.

TABLE 1.—*General Summary of Data—Continued*

Case, Age, Sex, Ear	First Seen / Operated On / Last Seen	Duration, Years	Cause	Family History of Deafness	Previous Treatment	Average of Three Preoperative Audiograms: Average of Three Final Audiograms 128	256	512	1024	2048	4096	8192	Bone Conduction	Tinnitus	Examination: Otoscopic	Nose and Throat	Sinuses	General	Diagnosis
19 30 F L	2/ 1/38 4/ 7/38 5/ 2/40	8	Colds; T & A	14 in three generations	Dilation; inflation; bougienage	27 20	32 15	37 20	47 33	28 32	45 33	63 58	Impaired; = AC	Absent	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 104/65; P. 64; B. M. R. —21; Wass. neg.; hypothyroid; allergic	Chronic catarrhal deafness
20 31 F R	1/10/38 4/ 8/38 5/27/40	4	Aviation	Negative	Dilation; inflation; bougienage	43 43	48 52	48 47	67 70	60 52	58 55	60 57	Impaired; = AC	Severe	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 116/80; P. 90; Wass. neg.	Chronic catarrhal deafness
21 33 M R	2/15/38 4/20/38 5/ 1/40	4	Fractured nose	Negative	Dilation; inflation; bougienage	5 5	10 0	15 8	43 38	47 45	53 47	48 47	Impaired; = AC	Absent	T+ R+ S LC—	Septum operation; T & A	Normal to roentgen examination	B. P. 112/78; Wass. neg.; P. 64	Chronic catarrhal deafness
21 33 M L	2/15/38 6/21/39 5/ 1/40	4	Fractured nose	Negative	Dilation; inflation; bougienage	5 7	17 0	20 8	42 40	52 52	50 65	57 53	Impaired; = AC	Absent	T+ R+ S LC—	Septum operation; T & A	Normal to roentgen examination	B. P. 112/78; Wass. neg.; P. 64	Chronic catarrhal deafness
22 37 M L	4/13/38 5/10/38 6/28/38	5	Chronic sinus disease	Negative	None	8 5	18 10	27 23	50 40	60 55	68 65	55 53	Impaired; = AC	Absent	T+ R+ S+ LC—	Normal; tonsils normal	Normal	B. P. 145/90; P. 88; Wass. neg.	Chronic catarrhal deafness
23 48 F R	5/21/38 4/31/38 12/11/39	36	Familial	Repeated 3 generations	Dilation; inflation; bougienage	37 40	38 45	42 53	57 58	58 35	57 42	33 33	Normal; = AC	Severe	T+ R+ S LC—	Septum operation; T & A	Normal	B. P. 114/80; P. 60; B. M. R. —20; Wass. neg.; X-ray: hyperostosis frontal bone	Chronic catarrhal deafness; otosclerosis(?)
24 40 F L	4/ 9/38 6/16/38 5/ 3/40	15	Postpartum	Negative	Dilation; inflation; bougienage; electrical therapy	45 43	48 43	47 48	53 57	40 42	40 37	27 23	Impaired; > AC	Present	T+ R++ S+ LC—	Normal; T & A	Normal	B. P. 112/80; P. 80; Wass. neg.; Hgb. 80%	Chronic catarrhal and nerve deafness
25 35 F R	6/30/38 7/14/38 1/27/40	20	Quinsy; colds	Negative	Dilation; inflation; bougienage	18 28	22 32	25 32	32 35	22 23	27 32	28 32	Normal; > AC	Postoperative	T R+ S+ LC—	Normal; T & A	Normal	B. P. 122/80; P. 96; B. M. R. 19; Wass. neg.; Hgb. 81%	Chronic catarrhal deafness; toxic goiter

26 30 F R	6/20/38 7/26/38 3/11/40	5	Colds	Negative	None	56 48	60 43	63 53	65 63	60 52	38 35	17 13	Impaired; = AC	Absent	T+ R+ S LC—	Normal; T & A	Normal	B. P. 122/85; Wass. neg.; B. M. R. —14; Hgb. 75%	Chronic catarrhal deafness; otosclerosis(?)
26 30 F L	6/20/38 7/25/39 3/11/40	5	Colds	Negative	None	60 30	70 57	68 55	75 70	55 65	38 32	20 17	Impaired; = AC	Absent	T+ R+ S+ LC—	Normal; T & A	Normal	B. P. 122/85; B. M. R. —14; Hgb. 75%	Chronic catarrhal otosclerosis(?)
27 37 F R	9/24/36 9/13/38 4/18/40	12 *	Colds	Negative	Nose and throat	45 30	47 37	52 38	62 57	53 42	63 65	60 60	Normal; > AC	Present	T+ R+ S+ LC—	Hypertrophied turbinate; T & A	Normal	B. P. 124/76; Wass. neg.	Chronic catarrhal deafness
28 27 F R	9/14/37 9/15/38 7/20/39	27	Birth	Negative	Dilation; inflation; bougienage	45 42	47 48	52 57	62 63	53 53	45 45	15 17	Impaired; > AC	Present	T R S LC+	Normal; T & A	Normal	B. P. 114/80; P. 88; Wass. neg.	Congenital absence of stapes
29 60 M L	10/17/38 11/ 1/38 6/12/39	4	C. C. O. M.	Negative	None	58 38	35 42	42 38	48 48	60 57	.. ..	.. ..	Impaired; < AC	Present	T+ R+ S+ LC—	Normal; T & A	Normal to roentgen examination	B. P. 124/70; Wass. neg.; Hgb. 85%	Chronic catarrhal deafness
30 44 M L	12/30/38 2/16/39 3/11/40	13	Allergy; colds	Brother;	Dilation; inflation; bougienage treatment for allergy	53 55	58 55	60 55	63 55	48 45	83 80	75 70	Impaired; = AC	Present	T+ R+ S+ LC—	Normal; T & A	Right antrum cloudy on roentgen examination	B. P. 122/80; Wass. neg.; P. 72	Chronic catarrhal deafness
31 12 M R	4/18/38 6/20/39 3/28/40	6	A. O. M.	Negative	None	58 48	60 57	55 47	48 48	50 40	53 48	70 58	Normal; > AC	Absent	T+ R++ S LC—	Normal; T & A	Normal	B. P. 112/74; Wass. neg.	Deafness following mastoid operation
32 43 F R	6/19/39 7/11/39 3/23/40	1½	Trauma	Negative	None	32 22	32 30	33 27	32 33	33 30	60 55	75 70	Normal; > AC	Severe	T+ R+ S+ LC+	Normal; T & A	Normal	B. P. 112/76; P. 76; B. M. R., —10; Wass. neg.; Hgb. 80%; low sugar tolerance; allergic	Chronic catarrhal deafness
33 34 F R	7/24/39 11/28/39 4/ 1/40	¼	Acoustic trauma	Negative	Dilation; inflation; bougienage	27 19	38 23	45 30	45 40	40 35	15 10	35 37	Impaired; = AC	Present	T+ R+ S LC—	Septum operation; T & A	Normal to roentgen examination	B. P. 124/80; Hgb. 75%; Wass. neg.	Chronic catarrhal deafness

observed over considerable periods. For the experimental animals in which serial sections of the temporal bones were made, eight weeks was the longest interval between operation and killing of the animal. In no instance was there apparent damage to the membrane. Had there been damage to the round widow membrane in these older clinical cases, there might reasonably be expected to be some disturbance relative to absorption of the intralabyrinthine fluid. Such a derangement would undoubtedly result in a more marked change than any observed, such as further impairment after three to four postoperative years. This phase of the subject is being investigated.

A statistical analysis of all but the most recent case, making a total of 35 operatively treated ears, has been prepared (table 1).

In these calculations the hearing level in all cases at one month intervals after operation has been averaged and the probable error calculated. These levels at different intervals can be compared with averages of three preoperative audiograms of all cases calculated in the same manner. The data are derived from 416 audiograms.

AUDIOMETRIC ANALYSIS OF OPERATIVE RESULTS

The greatest improvement in decibels observed at any postoperative period in any case for all frequencies is shown below. Any one of these decibel improvements is far beyond the observed variation in statistical analyses. It must be assumed, therefore, that these recorded improvements in hearing are the direct result of the particular surgical procedure employed. These figures are contrasted with the highest level in the preoperative period rather than with the lowest or the average of at least three. The final maximum observed improvement regardless of time for all patients and all frequencies is also shown. Again these figures are beyond any expected variation in repeated audiograms.

Frequency	128	256	512	1024	2048	4096	8192
Maximum improvement	25	25	30	35	30	25	25
Final maximum improvement	25	15	20	15	15	20	20

The tabulation presented has immediate significance for any one familiar with statistical data. It may be presented, however, in graphic form with a few simple explanations. In chart 1 the base lines represent the actual average hearing level in all cases for the specific frequency and its width, the calculated probable error, plus or minus, and the level from which improvement or impairment must be regarded as unqualifiedly positive or negative. In this particular tabulation are included data from all cases including 9, mentioned in the foregoing paragraphs, which are now regarded as completely unsuitable for operation. In other words, the operative results are presented in the most unfavorable manner possible for support of this operative procedure.

Against the base line with its probable error and spread is plotted the average level with the probable variation, the vertical line indicating spread. Overlapping of the base line by the composite audiograms must represent, from a statistical standpoint, a negative result. However, so long as the composite audiogram sustains a level above the base line it must be accepted as revealing an actual improvement in average

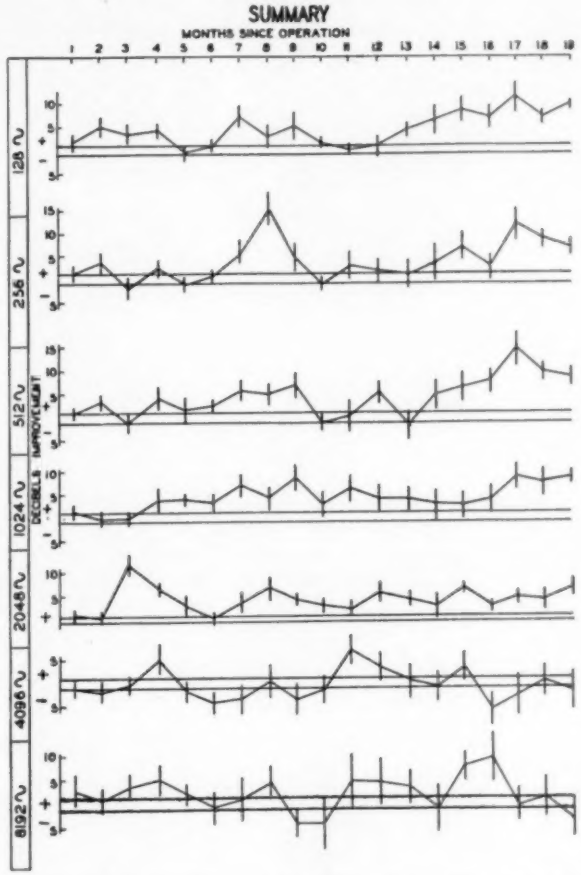

Chart 1.—Progress of improvement following operation. The double base line represents the grand average of the three preoperative audiograms made in each case and the range of probable error. Each point represents an average of audiograms made on all patients for the indicated month following operation. The one audiogram made on each patient during the month has contributed to this average. The short vertical lines show the range of the average as indicated by the probable error. Note that in the five lower frequencies the general level is preponderant above the control base line and never actually below it.

hearing for the 35 ears during the period of nineteen months. Unfortunately, sufficient material (not less than ten audiograms) is not available for periods greater than this.

In the report made two years ago,[2] discussion centered on the observed individual variation between successive audiograms. The present data have been prepared to meet this particular criticism. In chart 1 no sensational improvement is recorded. However, it must be recognized that in a nineteen month period following the round

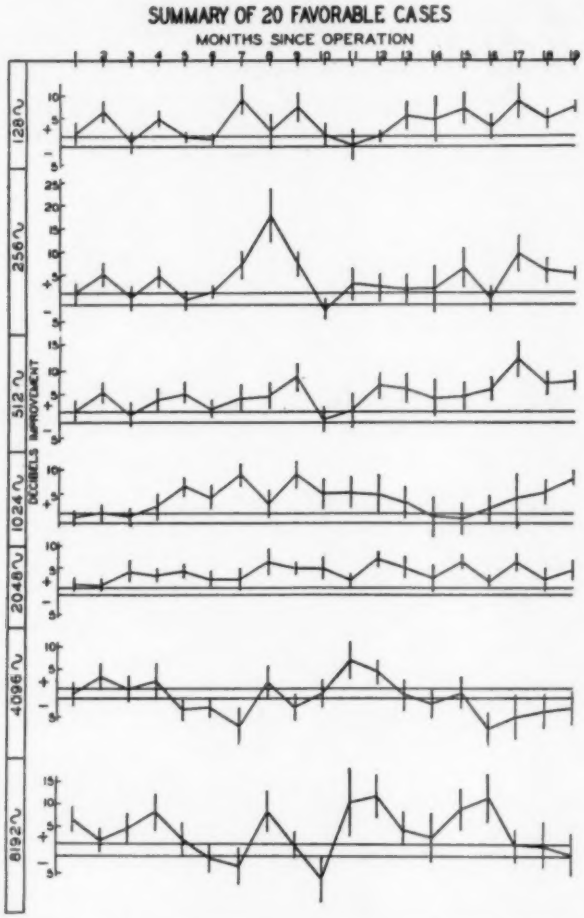

Chart 2.—Summary of twenty favorable cases. Although there is not a great difference in the recorded improvement between the two groups, reference to the corresponding tables will indicate that the base lines of the frequencies in the favorable group are at a higher level and that the spread of probable error is greater. The latter fact is due entirely to the smaller number of audiograms included in the analysis.

window graft operation there is for the frequencies 128 to 2048 a tendency toward improvement, if not actual improvement, in the hearing level.

It is also apparent that in the postoperative course the initial improvement of any real consequence develops between the sixth and the ninth month. For some undetermined reason this is followed by a decline,

TABLE 2.—*Summary—All Patients*

Frequency		128		256		512		1024		2048		4096		8192	
Month	Number of Audiograms	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error
*	106	37.8	1.0	41.2	1.0	45.9	0.9	50.9	0.9	47.8	0.7	53.6	1.1	51.2	1.2
1	35	36.3	1.8	40.3	1.8	45.0	1.6	49.7	1.4	47.0	1.4	54.7	2.1	48.6	2.7
2	29	32.9	2.1	38.6	2.3	42.4	1.9	51.7	1.7	47.4	1.5	55.5	2.3	50.9	2.4
3	27	34.5	2.0	43.2	2.2	47.2	2.0	51.2	1.6	35.9	2.3	53.0	2.0	47.6	3.0
4	18	34.7	1.8	38.1	1.9	41.7	2.1	47.0	2.3	41.4	1.3	48.6	3.0	40.8	3.8
5	21	38.8	1.4	42.4	1.7	44.5	2.0	46.9	1.6	45.0	2.1	55.5	2.1	49.8	3.2
6	22	36.8	1.5	40.7	1.6	44.3	1.5	47.8	1.9	47.5	1.6	57.7	2.2	53.9	3.5
7	15	30.3	2.5	35.3	2.4	41.0	2.1	44.0	2.1	44.0	2.0	57.0	3.1	50.3	4.6
8	15	35.0	2.6	25.7	3.4	42.0	2.3	47.7	2.6	41.0	2.5	53.0	3.6	46.7	3.5
9	14	32.2	3.3	36.4	3.2	40.0	2.9	42.5	2.7	43.6	1.3	57.1	3.0	55.4	3.3
10	13	36.2	1.7	42.3	1.7	46.9	1.9	48.0	2.7	44.6	1.7	55.0	2.9	55.4	5.9
11	9	37.8	2.9	38.3	3.1	45.6	3.4	45.6	2.9	45.6	1.6	46.7	3.6	36.7	6.0
12	14	36.8	2.1	39.3	2.7	41.4	2.6	46.8	2.9	41.8	2.2	50.0	3.0	46.8	4.8
13	13	33.5	2.7	40.4	2.9	47.7	2.9	46.9	2.8	43.5	1.7	53.1	3.2	47.7	3.6
14	10	31.5	3.7	37.5	4.1	41.0	3.3	49.0	3.4	44.5	2.4	54.5	3.1	52.5	5.1
15	11	29.0	3.1	34.1	3.6	40.5	3.1	49.5	2.8	40.9	1.4	50.0	3.3	43.2	3.6
16	8	31.9	2.5	38.8	2.8	38.1	2.5	46.9	2.8	45.0	1.4	59.4	3.5	41.3	5.2
17	7	26.4	3.9	29.3	4.0	31.4	4.0	42.1	2.7	42.9	1.9	56.4	4.8	51.4	3.9
18	11	30.9	1.7	33.6	2.2	36.3	2.0	43.2	3.2	43.6	2.2	53.2	3.4	50.0	4.5
19	12	28.3	1.3	34.6	1.5	37.5	2.0	42.1	1.7	41.7	1.9	55.4	4.1	54.6	4.4

* Average of three preoperative audiograms.

TABLE 3.—*Summary of Audiograms in Twenty Favorable Cases*

Frequency		128		256		512		1024		2048		4096		8192	
Month	Number of Audiograms	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error	Average	Probable Error
*	77	35.3	1.2	39.0	1.1	44.2	1.0	49.0	0.9	47.3	0.8	51.6	1.2	52.7	1.5
1	25	33.4	2.2	38.0	2.2	43.2	2.0	49.2	1.7	45.8	1.5	51.8	2.4	46.0	2.7
2	21	28.8	2.2	34.3	2.5	39.0	2.2	48.3	2.1	46.0	1.6	48.1	2.7	50.5	2.5
3	19	35.5	2.2	40.8	2.4	43.9	2.3	48.9	1.7	43.4	2.2	50.5	2.6	48.2	3.2
4	12	30.4	1.8	34.6	2.1	40.4	2.5	46.7	2.9	43.8	1.6	49.2	3.9	44.6	4.0
5	13	34.6	1.2	39.2	2.0	38.8	2.1	42.7	2.0	43.1	1.6	55.0	2.4	50.4	3.4
6	15	34.7	1.7	37.7	1.5	42.7	1.9	44.7	2.6	44.7	1.9	54.7	2.2	54.7	3.1
7	10	26.0	3.0	31.5	2.8	38.0	2.4	40.0	2.2	44.5	2.2	58.0	3.8	56.5	4.2
8	10	33.0	3.6	18.0	5.4	39.5	2.5	46.0	2.8	41.0	2.7	49.5	3.5	44.5	4.1
9	11	27.7	2.9	31.4	2.7	35.5	2.7	40.0	2.8	42.3	1.5	54.5	2.8	51.3	2.9
10	9	33.9	2.1	41.1	2.3	45.0	2.4	43.9	3.2	42.2	2.2	51.7	2.9	59.9	5.4
11	7	35.7	3.1	35.7	3.6	42.9	3.9	43.6	3.6	45.0	1.9	44.3	3.9	42.9	6.9
12	11	34.1	1.6	36.4	3.1	37.3	2.9	44.1	4.1	40.0	1.7	46.8	2.9	40.9	4.7
13	10	29.5	3.0	37.0	3.3	38.0	3.2	45.5	3.7	42.0	2.1	52.0	3.3	48.5	3.9
14	8	30.6	4.4	36.9	5.0	40.0	3.9	48.8	4.2	44.4	2.9	53.8	3.2	50.0	5.6
15	10	28.0	3.3	32.5	3.8	39.5	2.9	49.0	3.1	41.0	1.5	51.5	3.2	44.0	4.4
16	8	31.9	2.5	38.8	2.8	38.1	2.5	46.9	2.8	45.0	1.4	59.4	3.5	41.3	5.2
17	7	26.4	3.9	29.3	4.0	31.4	4.0	42.1	2.7	42.9	1.9	56.4	4.8	51.4	3.9
18	10	30.5	1.9	33.0	2.4	36.5	2.3	43.5	2.3	44.5	2.4	55.5	3.7	51.5	5.7
19	10	27.5	1.3	33.5	1.6	36.0	2.1	40.5	1.8	42.5	2.4	55.0	3.5	54.0	4.8

* Average of three preoperative audiograms.

with a subsequent elevation in the hearing level between thirteen and nineteen months. Except in frequencies 4096 and 8192, no impairment over the control level occurs.

A second statistical tabulation (table 2 and chart 2) presents data derived from all cases in the series which can now be accepted as favorable for operation, 9 of the total 30 being eliminated. The contrast in the level of this group and the preceding one is apparent though not marked.

On the basis of these data, fixation of the round window membrane with a tissue graft does produce an audiometric improvement in hearing. This fact is further supported by the following composite individual charts.

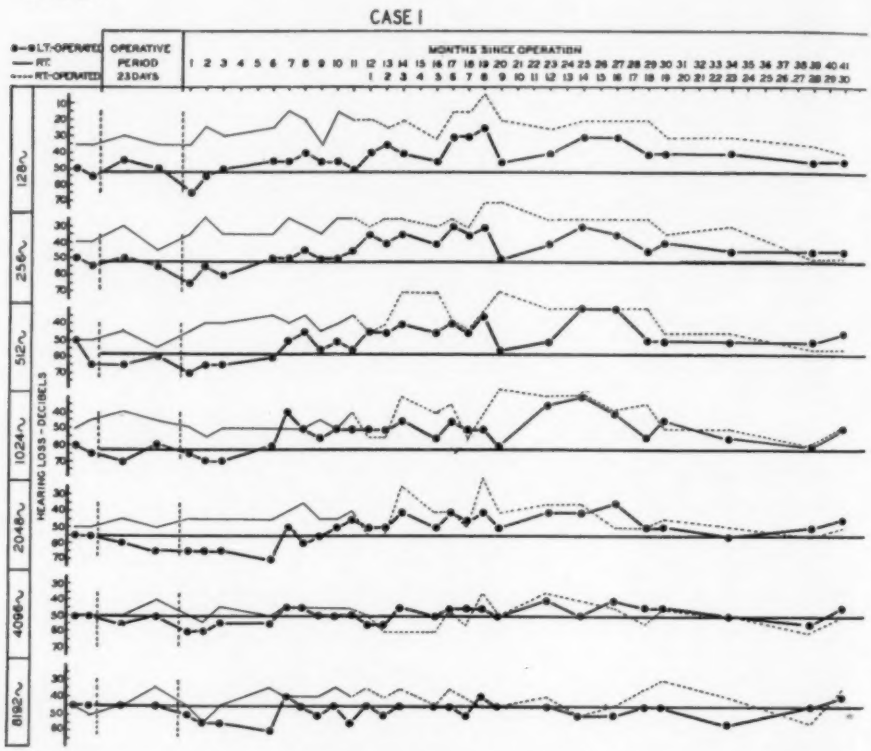

Chart 3 (case 1).—Twelve months after operation on the left ear the hearing level of the two ears was equal, and a second operation was performed. Improvement continued in both ears up to thirty-one and nineteen months respectively after the operation. A decline then set in, but at forty-one and thirty-one months respectively the level of hearing in the two ears was still above its preoperative level. In this case there was marked subjective improvement. (Charts 3 to 8 inclusive are composite audiograms in which each frequency is charted separately. The operative period represents the time between operation and complete healing of the ear drum.)

In chart 3 (case 1), representing the longest recorded period of observation, forty-one months, improvement has been maintained, though

not at the maximum level. The second operation was done when the level of hearing in the two ears was approximately the same, and, although there was improvement for eighteen months, this has subsequently declined.

In case 4 (chart 4), observed for forty months, the same situation holds. The maximum improvement has not been maintained, but there is still an increase over the preoperative level. In case 19 (chart 5), marked improvement was maintained in the operatively treated ear over

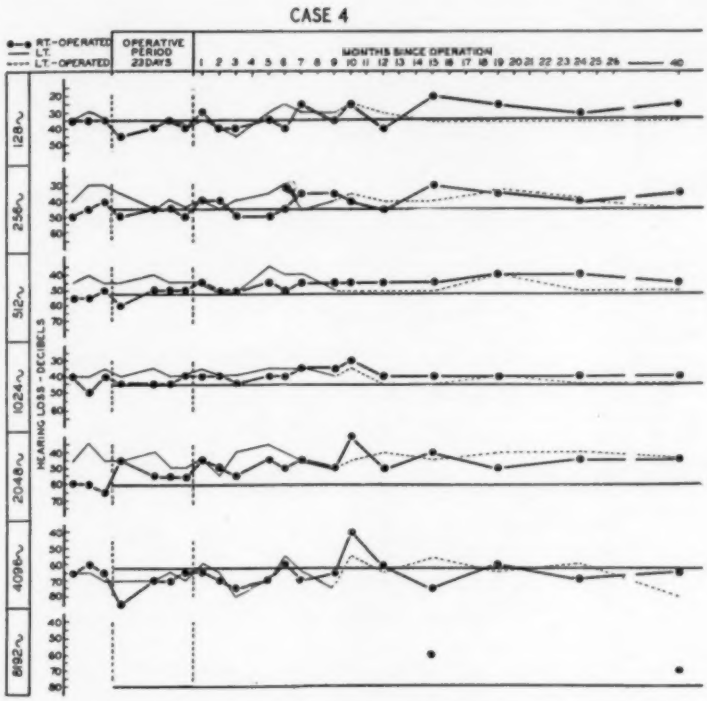

Chart 4 (case 4).—Records for 2 ears followed for forty and thirty months respectively. The audiometric improvement is not striking except for frequency 2048. There is marked subjective improvement.

a period of twenty-five months. In case 26 (chart 6), another bilateral operation was performed, with improvement in both ears for twenty and eight months respectively. In case 27 (chart 7), followed for nineteen months, there was a definite and sustained improvement. In case 33 (chart 8), the most recent case, there was a beginning improvement for all frequencies. This case is not included in any of the compilations, owing to the short period of observation. Each of the patients has had marked subjective improvement, and in 4 cases economic security has been reestablished.

SUBJECTIVE IMPROVEMENT

Subjective improvement is a matter of great interest and probably of prime importance. Obviously the 6 patients on whom a second operation was performed after a year's interval had sufficient subjective improvement to warrant their electing further surgical treatment. In the total group of 30 cases, 18 patients have reported subjective improvement, or 60 per cent. With elimination of the 9 unfavorable cases, there would appear subjective improvement in 85 per cent. A proper

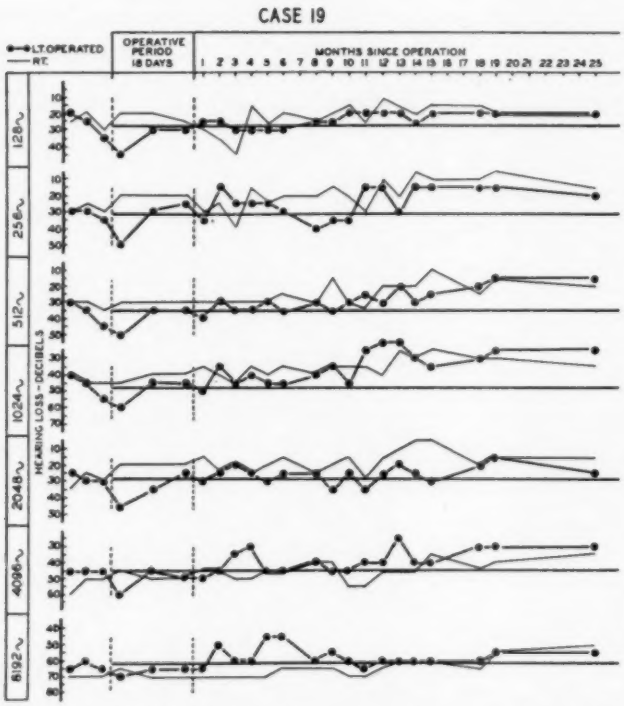

Chart 5 (case 19).—After twenty-five months the operatively treated ear is the better of the two. There is marked subjective improvement.

inquiry would be the extent or value of this reported improvement. Five patients have been able to continue their professional, business or scholastic activity when before operation the hearing loss was proving a serious handicap and a nervous and mental strain. The others, though not engaged in any gainful pursuit, are definitely relieved of the strain of deafness and are able to lead their lives in a relatively normal manner. One, a visiting nurse, now has no further need for a bone conduction hearing aid and is eager to have the second operation done. A young lawyer who experienced great difficulty in hearing the testimony of

witnesses and the judge's charge to the jury had operations performed on both ears and up to the present time has carried on his work with relatively little difficulty. A young woman occupying an important executive position in a large department store had bilateral operations and can now carry out her work with little of the nervous strain and fatigue she originally experienced. A schoolgirl having marked difficulty with her high school work was operated on twice in succeeding years. She finished high school and completed a course in a business college, where it was not known that she had any impairment of hearing.

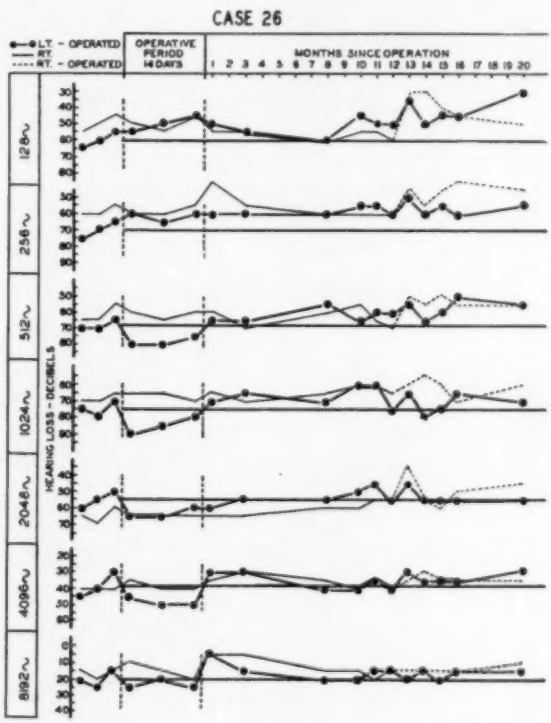

Chart 6 (case 26).—Bilateral operations followed twenty and eight months respectively. The audiometric improvement was not striking, but at the time of the second operation the originally poorer ear was the better of the two. Subjective improvement has been marked throughout, and there has been a definite rehabilitation despite the original low hearing level.

But this phase of the subject need not be elaborated further and is offered simply to satisfy the general insistence on the importance of subjective improvement regardless of the audiogram. It is still felt that the psychologic aspect of deafness makes subjective improvement a factor which must be appraised with considerable caution.

In a previous report [3] standards were set up which should be reached before actual improvement could be ascribed to any form of therapy, whether medical or surgical. Unfortunately no general data are available from which comparison may be drawn. In the individual case the results may be dramatic and gratifying, as has been shown by charts of selected operative results. It is felt that properly compiled statistical data are the only standard of efficacy of any given therapeutic measure.

Such data must be derived from repeated air conduction and bone conduction audiograms and loudness balance tests. Whether threshold

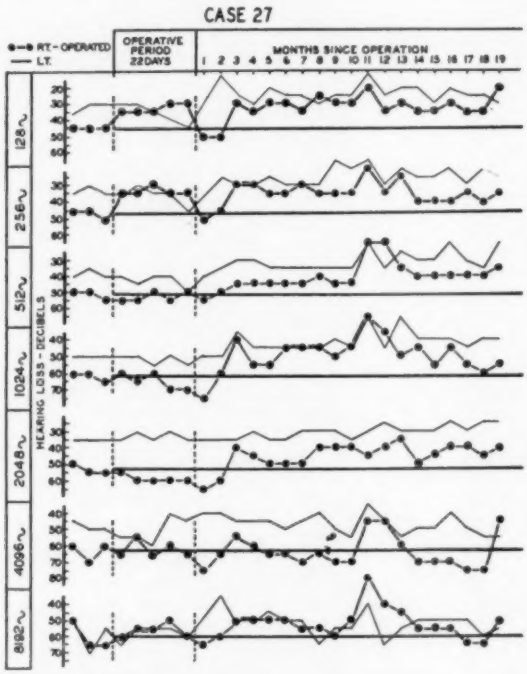

Chart 7 (case 27).—Unstable but continued improvement in the hearing level of the operatively treated ear for nineteen months. The patient has given up the use of a bone conduction hearing aid owing to the marked subjective improvement.

audiograms represent a true measure of hearing or not, they are the only means generally available for appraisal of therapeutic results. Certainly no increased impairment fails to record itself in the threshold audiogram, while the subjective improvement may not be a similar matter of audiometric record. The general feeling of impatience with threshold audiograms is largely due to the fact that such measurements do not fulfil an expected improvement level. Probably no individual frequency gain is of consequence unless it is at least an average of 10 decibels in repeated tests. Comparison of two individual audiograms is inconclusive.

At no level of hearing would a maintained gain of less than 10 decibels be of acoustic importance. Finally, the required duration of improvement has been set at five years. Such duration is quite arbitrary but is an entirely reasonable goal. In the present series this particular standard is as yet unachieved beyond the forty-one month interval.

The data from this four year period of observation are presented from every possible standpoint. The statistical analysis in all cases indicated

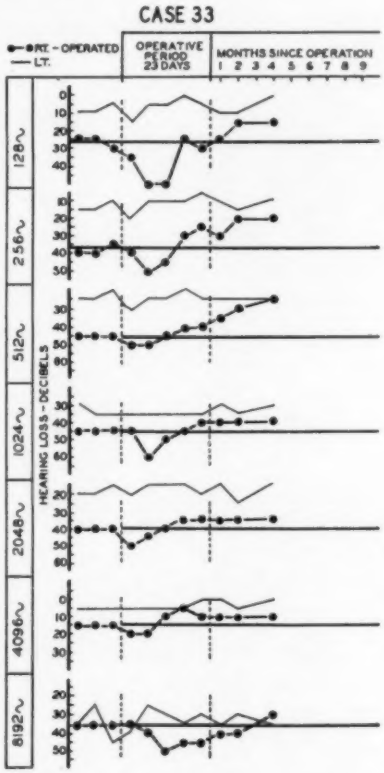

Chart 8 (case 33).—Record in the most recent case. The deafness was due to acoustic trauma. Note the beginning improvement for all frequencies at four months as noted in the average charts 1 and 2. Severe tinnitus in this case was partially relieved.

a trend toward improvement, despite the fact that there have been included not only favorable cases in which no improvement has occurred but cases in which the patients would not now be operated on under any circumstances and in which of course there was no improvement. These statistical data are final and complete. All factors have been taken into consideration and given due importance. The round window graft operation has proved of benefit on the basis of average improve-

ment, with recognition given to extremes of variation and expected error for 35 operatively treated ears. It is suggested that this particular type of analysis, painstaking and tedious as it may be, might well be used as the basis of appraisal of all types of therapy for deafness.

Composite audiograms showing relatively favorable results extending over varying periods have been presented, and in this connection it must be remembered that no patient's hearing has been impaired as a result of this surgical treatment.

SUMMARY

A tissue graft was placed in 36 ears. The longest period of observation was forty-one months, and the shortest, six months. No operative complication of any sort has resulted from this procedure.

In 1 case postoperative tinnitus developed. Preoperative tinnitus was reduced in 5 of 15 cases but was never cured.

No therapeutic measure, either medical or surgical, cures deafness. In 60 per cent of the series reported there has been a subjective improvement in hearing. Of the patients in the favorable cases, 9 being eliminated from the total, 85 per cent have experienced subjective benefit. This obviously emphasizes the importance of more rigorous restrictive standards for selection of operative cases.

A statistical analysis of all cases, whether favorable or unfavorable, in the light of this four years' experience demonstrates that the trend is toward an improvement in hearing. The operative results are reported in the most unfavorable manner for demonstration of the possible beneficial results occurring from the round window operation. Provided the patient is made to realize that no immediate improvement may be expected but that some relief may reasonably develop in six months and that future treatment, whether medical or surgical, is in no way jeopardized by the round window graft operation, it would seem that continuance of the procedure is justified in a few carefully selected cases of deafness which have failed to respond to universally applied local therapeutic measures.

My assistant, Miss Eva Thompson, prepared the running composite audiogram charts, which were first described and presented before the American Otological Society in 1938. Miss Thompson also made audiometric studies of air conduction and bone conduction and loudness balance tests.

My associate, E. G. Witting, Ph.D., developed special audiometers and refinements in audiometric technic, made suggestions for special procedures and for analysis of auditory acuity and intelligibility of speech and performed the difficult task of analyzing 416 audiograms.

ACUTE INFECTIONS OF THE EPIGLOTTIS

J. MACKENZIE BROWN, M.D.
LOS ANGELES

Acute inflammatory disease of the epiglottis is a clinical entity which is encountered rather infrequently. There are several types, which will be described.

REVIEW OF LITERATURE

The literature on abscesses and acute inflammations of the epiglottis is difficult to analyze because of the fact that many cases are recorded under the classification of "abscess of the larynx." In studying the case reports in most articles on laryngeal abscess, it becomes readily apparent that considerably more than one half of all such abscesses are epiglottidean.

Brown (1893) reported 2 cases of laryngeal abscess, in 1 of which puncture revealed a localized abscess of the epiglottis. He cited Mackenzie, who found involvement of either the epiglottis or the ary-epiglottic folds in 9 of 13 cases of laryngeal abscess.

Semon (1895) delivered an address in which he championed the etiologic and pathologic identity of all inflammatory diseases of the pharynx and of the neck. In this discussion he reported 1 case of suppurative epiglottitis in which the surrounding pharynx was not inflamed. He ascribed the infection to an original focus somewhere in the pharyngeal ring of lymphoid tissue (Waldeyer's ring).

Theisen (1900) made a valuable contribution to laryngologic progress by accurately describing the clinical picture of "acute infectious epiglottitis," a term he suggested in place of the previously used "angina epiglottidea anterior" described by Michel in 1878. (For an interesting and exhaustive survey of the early literature on this subject, Theisen's article should be consulted). In this paper Theisen stated, "At the very outset I will take the stand that true angina epiglottidea anterior (acute infectious epiglottitis) occurs quite often as a primary affection and that it is a separate and distinct condition." He reported 3 cases in this paper.

Later, Theisen (1914) reported several other cases of acute epiglottitis, but he completely reversed his former stand, retracted his statement

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of separate causation and expressed agreement with the earlier findings of Semon.

Lederman (1914) reported 4 cases of "acute phlegmonous epiglottiditis," which he described as an infectious process involving the submucous structures of the epiglottis and contiguous tissues, manifested by a sudden onset with distinct local manifestations. A seemingly normal person has a sharp pain in the throat, dysphagia and a sense of fulness and obstruction in the throat. The condition is rare in children. Edema of the epiglottis in all of his patients extended to the aryepiglottic folds. He quoted Gougenheim to the effect that "the respiratory difficulty is due to the immobility of the crico-arytenoid articulation and not to aspiration of the swollen ary-epiglottic folds."

Key (1916) reported the case of a 36 year old physician, in which bacteriologic study revealed Bacillus influenzae. It is significant that the condition occurred during an epidemic of influenza. Key stated that "in spite of all arguments as to whether angina epiglottidea anterior is a primary disease or not, it is an undeniable fact that a curious inflammation of the epiglottis in which there is remarkably little involvement of the rest of the throat does occur."

Ballin (1916) reported 3 cases of laryngeal abscess in each of which there was secondary edema of the epiglottis. In his discussion he stated that abscesses of the epiglottis are the most frequently encountered abscesses of the larynx. Most of these are unilateral (occurring especially on the left side) and originate from the lingual surface of the epiglottis, the aryepiglottic folds, and the piriform fossae.

Shambaugh (1920) reported a case of acute epiglottitis as "acute perichronditis of the epiglottis" in which the edema was confined to the lingual surface and was followed by a spontaneous discharge of pus.

Cohen (1925) reported an idiopathic epiglottic abscess without any other associated inflammation of the upper part of the respiratory tract. The case was that of a 50 year old woman in acute terminal diabetic coma.

Scal (1926) reported 3 cases of epiglottic abscess in adults.

Daily and Allen (1926) reported a case of acute epiglottitis in an 8 year old child, in which recovery followed an emergency tracheotomy.

Gilmore (1930) reported 1 case of epiglottic abscess in an adult in which recovery followed multiple punctures of the abscess. He stressed the importance, however, of the imminent danger of laryngeal obstruction in such a patient and the necessity for careful observation for possible tracheotomy.

Hyde and Ruchman (1931) reported a case of acute laryngitis in a 4 year old child in which epiglottic edema was present, necessitating a tracheotomy.

Howard (1931), in an interesting report of 3 cases of laryngeal abscess, stated that in all 3 the epiglottis was the primary seat of suppuration, accompanied by edema of the aryepiglottic folds and the arytenoid cartilages. He mentioned also the fact that the first case on record was that of Roland, of Parma, in the thirteenth century.

Weiss (1932) reported 1 case of primary abscess of the epiglottis which responded to multiple punctures. He isolated Staphylococcus albus as the etiologic organism.

Tawse (1932) reported 3 cases of acute streptococcic ulceration of the epiglottis, in which antistreptococcic (antiscarlatinal) serum was used successfully in treatment. These cases probably would fit into Michel's classification of angina epiglottidea anterior.

Rebattu, Mounier-Kuhn and Millet (1935) reported the fatal case of a syphilitic adult, in which there was an epiglottic abscess associated with what was apparently a parapharyngeal phlegmon. Death was due to sudden laryngeal obstruction.

Noone and Shields (1937) reported a case of epiglottic abscess in an 11 month old infant, in which the abscess was incised under direct laryngoscopy, with a favorable outcome.

Coates, Shuster and Gordon (1938) reported 2 cases of epiglottic abscess in young adults, with recovery.

From an analysis of Lewis' excellent monograph on the last illness of George Washington, I feel, with others, that Washington's death may have been due to an abscess of the epiglottis.

ANATOMY

The epiglottis is a leaf-shaped structure consisting of a thin fibrocartilage, one of the three unpaired laryngeal cartilages. It has a free extremity, which is smooth, broad and rounded. The long, narrow stem is attached anteriorly to two structures; (1) the thyroepiglottic ligament, which is connected to the angle of the thyroid cartilage immediately below the superior thyroid notch, and (2) the hyo-epiglottic ligament, which connects the epiglottis with the upper border of the body of the hyoid bone.

Mucous membrane covers the epiglottis on both its surfaces, the anterior, or lingual, and the posterior, or laryngeal.

An elevation on its lower laryngeal surface is called the tubercle or cushion.

The lingual surface and the upper one half of the laryngeal surface are covered with stratified squamous epithelium. The lower one half of the laryngeal surface, near the base of the epiglottis, is covered with ciliated, pseudostratified columnar epithelium. The principal blood supply to the epiglottis is from the superior laryngeal artery, a branch

of the superior thyroid artery. The internal laryngeal nerve, a division of the superior laryngeal branch of the vagus nerve, supplies most of the mucous membrane of the epiglottis.

The lymphatic drainage of the larynx is divided into two principal channels, a supraglottic and an infraglottic. The epiglottic mucosa is

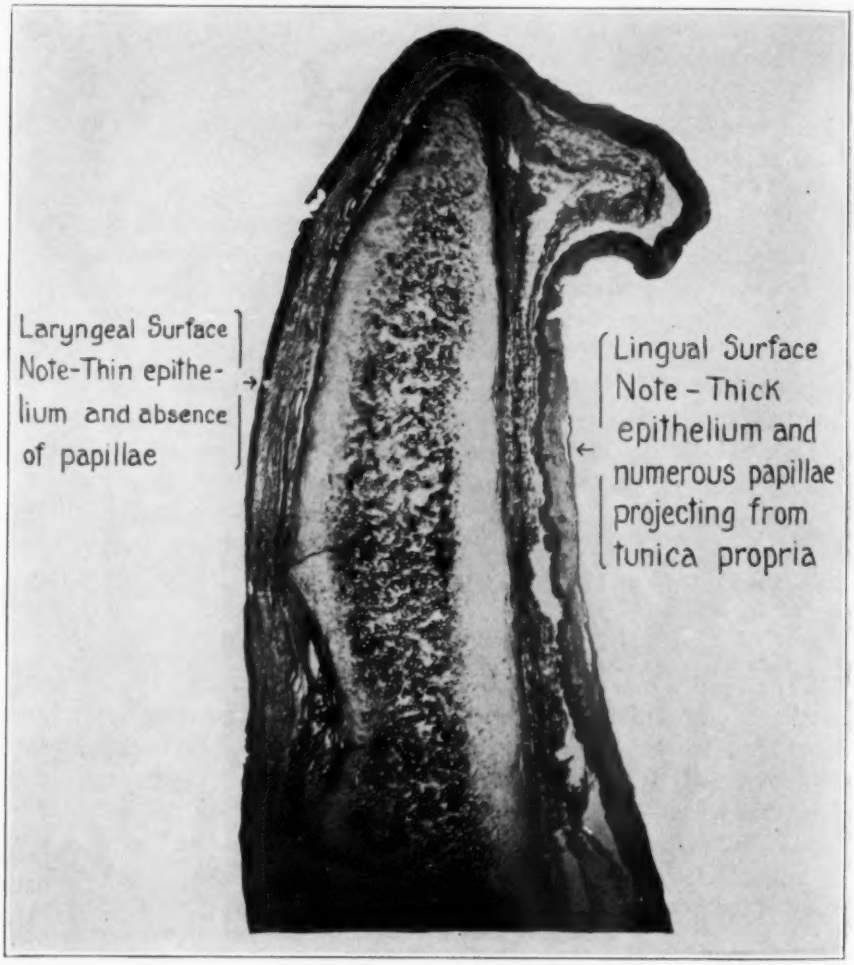
Laryngeal Surface
Note-Thin epithelium and absence of papillae
Lingual Surface
Note - Thick epithelium and numerous papillae projecting from tunica propria

Fig. 1.—Low power photomicrograph of a section of a normal human epiglottis.

drained by the supraglottic lymphatic vessels. These vessels accompany the superior laryngeal artery, pierce the hyothyroid membrane and end in the deep cervical lymph glands situated near the bifurcation of the common carotid artery.

Histologic studies by Hajek were applied to the pathogenesis of epiglottic infection in the following manner. He showed that the submucosa of the lingual surface is abundant and the mucosa not adherent, whereas on the laryngeal surface the mucous membrane is tightly adherent to the perichondrium of the epiglottic cartilage, with practically no loose areolar submucosa except in the region of the cushion.

As a result of Hajek's work it has been generally accepted that suppuration is confined to the anterior surface of the epiglottis.

However, recent studies in the department of anatomy of the University of Southern California have demonstrated significant differences in the histologic character of the epiglottis. It was noted that the stratified squamous epithelium on the lingual surface is three to five times as thick as it is on the laryngeal surface. Also, there are numerous papillae of connective tissue projecting from the tunica propria on the lingual surface, but there are none on the laryngeal surface. The connective tissue on the tunica propria is much more delicate on the laryngeal surface, containing less collagen but more elastic fibers. Taste buds were present, strangely enough, on the laryngeal surface, with none on the lingual surface.

It was noted that the tunica propria of the lingual surface is more tightly adherent and more dense than that of the laryngeal surface and that the tunica propria of the laryngeal surface is a loose and delicate structure. This can be seen in both human and animal tissue.

Accordingly, Hajek's view cannot be accepted entirely, as I have seen 2 cases of abscess of the epiglottis on the laryngeal surface. The histologic evidence would tend to substantiate the fact that suppuration may occur as frequently on the laryngeal as on the lingual surface.

PHYSIOLOGY

Functionally, the epiglottis, traditionally a very important factor in maintenance of body economy, having the supposed duty of protecting the larynx from the inspiration of food, drink and foreign bodies, is now known to be less important physiologically. Closure of the glottic chink is performed by the powerful action of the constrictor muscles of the larynx and not by any valvelike function of the epiglottis. Epiglottidectomy for carcinoma or tuberculosis is not followed by any serious consequences as far as laryngeal inspiration of food is concerned.

The epiglottis does, however, have a function, namely, bisection of a bolus of food or a volume of liquid into two channels, each directed to the right or to the left piriform fossa.

ETIOLOGY

The causation of acute epiglottitis is varied. Many authors have contended that the disease can be and most usually is a primary infection

unrelated to the rest of the upper part of the respiratory tract. In 1895, Semon reported 1 case of suppurative epiglottitis in which there was no apparent associated pharyngitis. Semon, however, ascribed the condition to the lymphoid tissue in some part of Waldeyer's ring. He stated that "the various forms of acute septic inflammation of the throat and neck, hitherto considered as so many essentially different diseases, are in reality pathologically identical, that they merely represent degrees varying in virulence of one and the same process."

According to Scal, "the most probable cause is a slight injury occurring at the base of the tongue while eating, or spreading from the

Fig. 2.—Indirect laryngoscopic (mirror) view of the epiglottis and the glottic chink in a case of acute ulcerative inflammation of the epiglottis (streptococcic).

lingual surface of the epiglottis to the root of the tongue on the aryepiglottic folds."

Jackson classified epiglottitis under the heading of "traumatic laryngitis" and listed various corrosives and thermal agents as etiologic factors.

Morrison stated that abscess of the epiglottis is most commonly associated with perichrondritis and mentioned radiation necrosis as a factor.

Thomson classified epiglottitis into primary conditions due to trauma and secondary conditions following acute tonsillitis, pharyngitis or lingual tonsillitis.

The cause of acute inflammation of the epiglottis has been considered by many authorities to be primary. However, from my experience, I

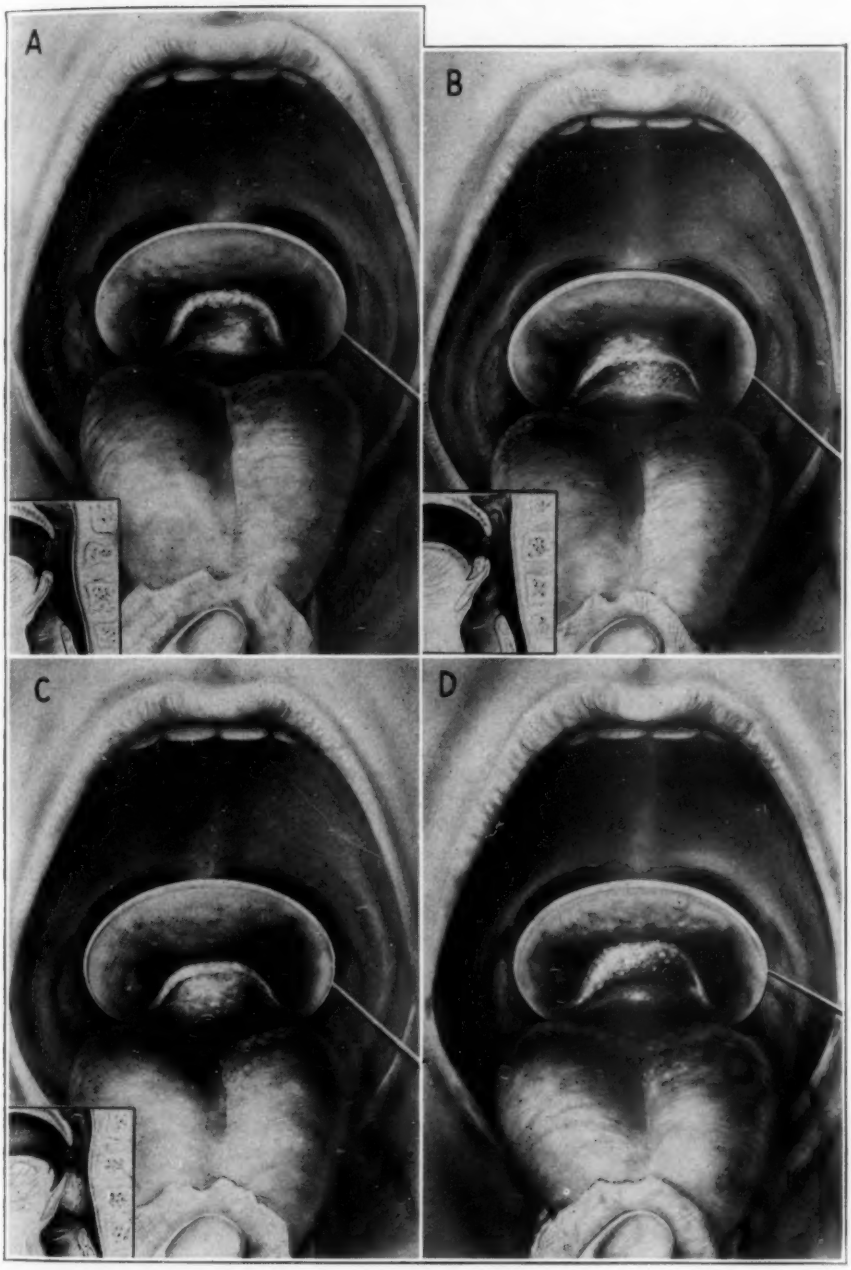

Fig. 3.—*A*, mirror view in a case of acute diffuse inflammation. *B*, mirror view in a case of abscess of the anterior (lingual) surface of the epiglottis. *C*, mirror view in a case of abscess of the posterior (laryngeal) surface of the epiglottis. *D*, mirror view in a case of acute infection of the epiglottis, with abscess formation on the lip of the organ. In the drawings the insert shows the sagittal section.

agree with the larger number of writers, who hold that the condition is always secondary to adjacent infections or trauma.

The bacteriologic picture of acute epiglottitis is the same as that of acute pharyngitis. The usual respiratory organisms, either alone or mixed, have been isolated in most cases.

DIAGNOSIS

The onset of acute epiglottitis is characterized by rapid development of a sore throat, painful swallowing and a thickened and muffled voice. Attempts to swallow may be followed by coughing and choking, and frequently the patient tends to hold the head and neck rigidly. The patient often complains of a lump in the throat on swallowing. Dyspnea occurs in direct proportion to the development of obstructive swelling.

The diagnosis can be made only on visualization of the epiglottis. This is done simply and effectively by indirect laryngoscopy.

From such examination, the following clinical types have been observed:

1. Acute diffuse inflammation.
2. Acute inflammation with abscess formation.
   (*a*) Abscess on the laryngeal surface.
   (*b*) Abscess on the lingual surface.
   (*c*) Abscess on the lip.
3. Acute ulcerative inflammation.

TREATMENT

If the condition is seen in the early stage, when there is no definite abscess formation and no marked tumefaction, conservative therapy may cause resolution. Such therapy consists of local and general supportive measures combined with chemotherapy. If the swelling increases or if there is clinical evidence of abscess formation, then under direct laryngoscopy the most prominent part of the swelling or any pointing area should be incised. Tracheotomy may become necessary if marked laryngeal obstruction occurs.

REPORT OF CASES

CASE 1.—On June 1, 1915, I was called to the country to see A. B., a white man aged 30. The patient was unable to speak and was having difficulty in breathing. Examination with a miror showed that the epiglottis was inflamed throughout, with a definite swelling on the posterior surface.

The swollen epiglottis was immediately incised at its most prominent point with a curved tonsil knife. A considerable amount of frank pus was obtained, and the patient was relieved of his dyspnea at once.

The following history was then obtained: One week previously he had had a sore throat, which lasted two days. He was well until thirty-six hours before I saw him. At this time he experienced a sudden pain in the throat, which continued, with the development of dyspnea and aphonia in the last six hours.

An uneventful recovery followed.

CASE 2.—G. W., a white woman aged 54, was admitted to the Los Angeles County Hospital with the complaint of sudden onset of sore throat two days previously and difficulty in swallowing for one day. The temperature was 101 F.

Examination revealed acute tonsillitis, laryngitis and a very edematous epiglottis, especially on the posterior surface.

In spite of conservative treatment, the swelling on the posterior surface of the epiglottis increased. The swelling therefore was incised under direct laryngoscopic vision, with release of a large amount of bloody pus. The patient immediately improved, and recovery was uneventful.

CASE 3.—M. C., a white man aged 26, was seen in the clinic of the Los Angeles County Hospital on April 9, 1939, complaining of a progressively severe sore throat of two days' duration and of pain in the left side of the neck.

Examination revealed acute pharyngitis, with an enlarged gland on the left side of the neck. No laryngoscopic examination was made at this time. On the following morning, examination in the department of otolaryngology disclosed a marked swelling on the anterior surface of the epiglottis, with a softened yellowish area at one point. Under direct laryngoscopic vision this yellowish area was opened, and a considerable amount of pus was obtained, culture of which showed Streptococcus viridans. The patient made an uneventful recovery.

CASE 4.—A. M., a Negro aged 50, was admitted to the department of otolaryngology of the Los Angeles County Hospital on Aug. 14, 1938. He gave the history of a sudden onset of dyspnea at 1 p. m., nine hours before admission. At 3 p. m. the dyspnea became severe enough to make him seek medical attention. On admission the patient showed considerable dyspnea. The temperature was 102 F. Attempts at laryngoscopic examination were unsuccessful, as the patient refused to cooperate.

Owing to the obvious evidence of laryngeal obstruction, a tracheotomy was advised, which the patient refused and for which no consent was available. He was given conservative treatment, and the dyspnea seemed to improve. At 1 a. m. he jumped out of bed, ran into the ward and dropped dead. An immediate tracheotomy and intracardiac administration of epinephrine were ineffective.

The diagnoses at autopsy were abscess of the epiglottis with acute edema of the larynx and acute pulmonary edema.

The autopsy protocol contained the following notes: "The anterior surface of the epiglottis is markedly edematous, is enlarged to three times normal and is fluctuant on palpation. The cut surface is reddened, and the fluctuant area is filled with thick green pus. The aryepiglottic folds are edematous, as are the true and false cords. The posterior surface of the epiglottis and the mucosae of the larynx and trachea are only slightly edematous and injected."

CASE 5.—D. C., a white man aged 29, had a sore throat on March 4, 1940. Four days later he became acutely ill and had a severe chill, with a temperature of 104.4 F. He was admitted to the Los Angeles County Hospital on March 8.

Examination at that time revealed an acutely inflamed pharynx, the mucous membranes of which were edematous and covered with a gray membrane which

extended over the tonsillar fossae and the soft palate. There was a group of enlarged glands in each side of the neck. The epiglottis was markedly edematous and covered with patchy ulcers consisting of necrotic areas covered with gray exudate and areas of hemorrhage. The same ulcerative process was present to a lesser extent on the arytenoid cartilages. The glottic chink was well open, however, and the cords were only mildly injected. The white blood cell count on admission was 17,200 per cubic millimeter; the value for hemoglobin was 96 per cent. He was given sulfanilamide therapy. He continued to complain bitterly about his throat, soon becoming comatose and delirious. On the following day, culture of material from the throat showed Str. viridans and clinical evidence of pneumonia of the lower lobe of the left lung. The patient was given immunotransfusions. At no time was there a blood culture yielding bacteria. The throat and chest gradually improved, and the patient had a slow but uneventful recovery and was discharged on March 25.

CASE 6.—R. S., a white woman aged 56, was seen at my office on Feb. 28, 1940, with the complaint of a severe sore throat of five days' duration, not relieved by ordinary remedies, and great difficulty in swallowing during the past twenty-four hours.

Examination showed the tonsils and pharynx to be acutely inflamed, with diffuse edematous inflammation of the whole epiglottis. The larynx proper was not involved. The patient was given conservative therapy and rest in bed. The edematous inflammation of the epiglottis subsided within one week.

CASE 7.—On Jan. 12, 1937, M. G., a white man aged 41, complained of a sore throat and hoarseness of two days' duration. The onset was sudden.

Examination revealed the epiglottis to be greatly swollen and injected. The greatest swelling was on the right half of the lip. The larynx could not be seen because of this swelling. The temperature was 100 F.

Conservative treatment with pharyngeal irrigations and sulfanilamide was instituted. At the end of two days the swelling appeared fluctuant.

Under direct laryngoscopic vision the right half of the lip of the epiglottis was incised, and a large amount of pus was obtained from an abscess. An uneventful recovery ensued within one week.

CONCLUSIONS

1. The epiglottis may be the site of localized infection or of abscess formation without involvement of the adjacent pharynx or larynx.

2. Such infections may be primary, due to trauma or secondary to adjacent infection.

3. Such infections of the epiglottis have their own peculiar and fairly constant symptoms and findings.

4. In my experience, an abscess may occur as readily on the laryngeal as on the lingual surface, a fact substantiated by recent histologic study.

5. If the foregoing facts are realized, earlier diagnosis and proper treatment may be employed to prevent serious complications.

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OSTEOMA OF THE MASTOID

REPORT OF TWO CASES

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MEMPHIS, TENN.

Osteomas of the mastoid are of so much interest on account of their rarity and their deforming possibilities and as etiologic factors in complications rising from the growths themselves as well as from surgical intervention that it seems to me that all cases should be described with a detailed account of the symptoms, the complications observed before, during or after any operative procedure and the final result.

There are three distinct types of osteoma of the mastoid: first, the firmly attached, nonmovable type; second, the movable type, which some times has a pseudarthrosis (both of these are alive); and a third type, small, hard, alive, movable and unattached, of which there are only 2 cases—Amberg's, which is contained in the literature, and my own case 2, which I am reporting in this paper. In Amberg's case there was possibly an attached pedicle; in my case there was no pedicle, but the tumor was slightly roughened on one side, from which area there was doubtless some vascular supply.

Marx,[1] in discussing exostosis of the external auditory meatus as a result of breaking off or degeneration of the pedicle, stated that there may be movable or free osteomas.

No case of dead or loose osteoma of the mastoid similar to those of the ethmoid and frontal sinuses reported by Tillmanns[2] has been found.

Several years ago, while doing an operation on the frontal sinus of a fresh cadaver, I found an osteoma the size of a small hazelnut in a left frontal sinus. The growth was lying perfectly loose in the sinus. There was no fluid or sign of infection of any kind in the frontal sinus.

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Read before the Section on Laryngology, Otology and Rhinology at the Ninety-First Annual Session of the American Medical Association, New York, June 12, 1940.

1. Marx, H.: Die Geschwülste des Ohres, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, pp. 490-568.

2. Tillmanns, H.: Ueber todte Osteoma der Nasen- und Stirnhöhle, Arch. f. klin. Chir. **32**:677-690, 1885.

The osteoma was very hard, with a smooth surface but slightly roughened on one side. No histologic examination was made.

A large percentage of osteomas of the mastoid are firmly attached to the underlying bone, with a rather broad base; a smaller percentage of these tumors are pedunculated.

Osteomas of the slightly movable type are situated usually in a depression of the mastoid bone with an intervening mass of cartilage or connective tissue. An exception occurred in Smith's case, in which there was an intervening layer of skin instead. Osteomas of this second type may be pedunculated also.

Of course, an osteoma may be of any shape and may arise in any position. The external auditory meatus may be encroached on or not, depending entirely on the point of origin and on the direction of growth.

In removing tumors of the fixed type, rather a usual occurrence is uncovering of the dura of the posterior fossa, the middle fossa or a lateral sinus, depending on the position of the tumor.

In my perusal of the literature I have found no mention of recurrence of an osteoma of the mastoid, but it seems to me that as complete a removal of the tumor as is practical, including its point of origin, should be carried out. Recurrence of osteomas of the accessory sinuses has been noted.

Dolbeau [3] was the first to describe an osteoma of the frontal sinus which had become completely detached from its base and was lying as a necrotic bony ball in the frontal sinus, similar to a urinary calculus.

Tillmanns [2] stated that Otto and Holmes Coote found loose osteomas in the frontal sinus. The skulls in which these osteomas were found, said Tillmanns, belonged to Brichet [4] who placed them in a museum at Dupuytren.

Eckert Möbius [5] mentioned 15 loose or dead osteomas of the nasal accessory sinuses, with the following points of origin:

Antrum of Highmore (Lederhose), 1 case.

Frontal sinus (Imre, Lediard, Kikuzi), 3 cases.

Ethmoid bone (all other authors), 11 cases.

He also said that an osteoma probably may become extruded spontaneously when small or may remain as a foreign body when large.

It seems to me that the most plausible reason for separation of an osteoma from its attachment is either pressure atrophy or suppuration.

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3. Dolbeau, H. F.: Exostoses du sinus frontal, Bull. Acad. de méd., Paris, 1886, vol. 21.

4. Brichet: Rapport, Bull. Acad. de méd., Paris **36**:564-599, 1871; Gaz. d. hôp. no. 54 ff.

5. Eckert Möbius, A.: Gutartige Geschwülste der innern Nase und ihrer Nebenhöhlen, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 5, pp. 107-201.

Tumors with wide bases, like most osteomas of the mastoid, would never become loose or dead, but those with small pedicles, such as have been described as existing in the accessory sinuses, might become both loose and dead.

I believe that in my second case there never was any attachment of the bony tumor to the mastoid bone. This tumor, I think, was an entirely different sort from those described by Tillmanns [2] as dead or loose osteomas of the sinuses. Campbell [6] has described a similar condition in the knee joint.

A few weeks ago I opened a frontal sinus for a chronic infection with a roentgen picture indicating an osteoma of this cavity and found two loose, distinct living osteomas.

There seems to be considerable diversity of opinion as to the origin of osteomas.

Kauffman,[7] in discussing the genesis of osteomas, said that two types are differentiated: (*a*) cartilaginous or epiphysial and (*b*) connective tissue preformed, that is, periosteal growths and so-called tendinous exostoses.

Tillmanns [2] and Dolbeau [3] stated that these tumors originate from embryonal cartilage.

Fetissof [8] stated that Arnold was the originator of the theory of development of osteomas from cartilage split off at an early stage of its development. In spite of its seeming fitness from the clinical standpoint, however, Arnold's theory does not have the histologic points in its favor. Moreover, if osteomas were developed from cartilage one would have to deal in the clinic with osteochondromas of the nose. However, among the great number of osteomas, the only osteochondroma described [8] originated from the ethmoid (case of Moure and Nadal).

In giving the results of microscopic examination in his cases Fetissof said that osteoblastic ossificatory process plays a very small part in the osteoma as compared with the formation of bone through metaplasia from the fibrous tissue. The fibrous tissue inside the osteoma is in its turn a direct continuation of the periosteum of the surrounding bone. The spongiose parts of the osteoma should be considered its younger parts. Furthermore, the compact bone is formed from the spongiose bone.

Campbell [9] stated that he knows no reason why an osteoma in or about the head could not arise from preosseous connective tissue, just

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6. Campbell, W. C., and Mitchell, J. I.: Loose Bodies in Joints, Indianapolis M. J. **32**:521-525, 1929.

7. Kauffman, E.: Pathology, translated by S. P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 2, p. 1195.

8. Fetissof, A. G.: Pathogenesis of Osteomas of the Nasal Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **38**:404-412, 1929.

9. Campbell, W. C.: Personal communication to the author.

as the normal bones of the head and face form in the embryo without interposition of cartilage.

It has often been stated that trauma and infection may have some bearing on the formation of osteomas, but it seems to me that they have little, if anything, to do with formation of osteomas of the mastoid, especially since in a study of the literature I found not a single case in which injury was mentioned in the previous history. Of the entire group of 30 cases there was a history of chronic suppuration in only 9.

From a microscopic standpoint osteomas are usually divided into three types: first, osteomas made up of very hard bone (osteoma eburneum); second, osteomas composed of spongy bone (osteoma spongiosum), and third, osteomas composed of both the eburnated and the spongy type (osteoma mixtum).

Lillie and Williams,[10] Vail,[11] Aprile [12] and Banyay and Janota,[13] as well as others, have reported on osteomas or exostoses of the external auditory meatus. Many of these were similar in structure, if not exactly identical, to those found in the mastoid proper, but since this paper deals primarily with osteomas of the mastoid I shall not attempt to abstract the cases reported by these authors.

In a rather extensive search of the literature only 30 cases of osteoma of the mastoid proper have been found. In the first 21 cases the growths were of the nonmovable type; in the next 9 cases they were of the slightly movable type, and the last case was Amberg's, in which the growth had a type of its own, being entirely detached.

There follow abstracts of the 21 cases of nonmovable osteoma.

REVIEW OF REPORTED CASES

Paulian and Popp [14] reported the case of a man 43 years old with a history of illness extending back five years. The roentgenogram of the right ear showed a small exostosis of the mastoid at the sagittal suture, and there was also observed a small enostosis of the internal lamina of the cranium. At the level of the right petrous pyramid, just above the right semicircular canal, there was another osteoma, the size of a bean, and on the left mastoid process there was an enostosis symmetric to that on the right.

The enostosis situated on the right at the level of the petrous pyramid, by compression on the superior semicircular canal and the consecutive labyrinthine

10. Lillie, H. I., and Williams, H. L., Jr.: Osteoma of the External Auditory Canal, S. Clin. North America **11**:801, 1931.

11. Vail, H. H.: Case of Multiple Osteomata of Both External Auditory Canals, J. Med. **9**:28-31, 1928.

12. Aprile, V.: Fibroangioma, osteoma, papilloma del condotto uditivo, Rinasc. med. **10**:[300]-301, 1933.

13. Banyay, J., and Janota, F.: Ein mandelförmiges obturierendes Osteom des äusseren Gehörganges, Monatschr. f. Ohrenh. **67**:983-984, 1933.

14. Paulian, D. E., and Popp, L.: Small Osteoma of the Right Petrous Pyramid as Cause of Right Labyrinthine Vertigo, Spitalul **16**:427-429, 1936.

hemorrhage on the right, can explain the labyrinthine disturbance of Ménière's vertigo and labyrinthine deafness. The slight paresis of the right facial nerve may be similarly explained by slight compression of the nerve.

Burger [15] reported the case of a man 26 years old with a tumor of the right mastoid process which obstructed the external auditory canal, rather markedly affecting the hearing. The patient was not seen again until four years later, when he complained of pain in his right ear, which soon began to have a purulent discharge.

A painless swelling about the size of a cherry, red and fluctuating, developed below the right ear. Roentgen examination at this time showed a decided increase in the bony growth. In a few days the soft swelling below the ear was incised; bloody purulent fluid was evacuated, and a diagnosis of suppurative fistula of the bronchial canal was made, which was not substantiated.

Two weeks later the osteoma was removed with a chisel with the patient under ether anesthesia. In the removal normal mastoid cells were opened. The growth was larger than a walnut and was attached with a wide base. During the removal a large opening was made in the middle fossa and in the posterior walls of the external auditory meatus.

Microscopic examination showed a mass consisting of bony tissue with a small number of haversian canals in which there was practically no tissue. The diagnosis was eburnated osteoma.

Preusse [16] discussed osteoma of the mastoid process and reported a case. His patient had had an elevation behind the right ear for four months, which gradually increased in size until it was as big as half a walnut. In the four months prior to his examination by Preusse the patient had pains above and behind the right ear, deep headaches over the right temple, waking at night and a sense of pressure. The hearing was normal.

The growth was of the right mastoid; it pushed the auricle slightly forward. The tumor was uniformly rounded. The external auditory meatus was normal. An operation was done with the region under local anesthesia, with an incision made as for a mastoidectomy. The bony tumor was slightly nodulated and protruded into the mastoid process from the depths of the petrous bone. In the posterior and superior regions it reached almost as far as the parietal fissure. The tumor was removed with a chisel, several mastoid cells being exposed in the removal. The deeper parts of the tumor were removed bit by bit. A hemorrhage from the small vein leading to the sinus was checked by covering with a bit of muscle.

The histologic report revealed compact bone well supplied with blood vessels.

Buck [17] briefly reported a case reported to him in turn by Vandervoort. (Preusse said that this is the earliest known case of osteoma of the mastoid.) A woman 54 years old had an exostosis protruding from the central portion of the mastoid process, half the size of a nutmeg. It was hemispheric, smooth, painless and of more than thirty years' standing. The patient had had scarlet

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15. Burger, H.: Inneres Osteom des Warzenfortsatzes, Monatschr. f. Ohrenh. **65**:1447-1450, 1931.

16. Preusse, K. H.: Ueber Osteombildung am Warzenfortsatz des Felsenbeins, Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **31**:203-315, 1934.

17. Buck, A. H.: Diseases of the Mastoid Process, Their Diagnosis, Pathology and Treatment, Arch. Ophth. & Otol. **3**:173, 1873; translated, Arch. f. Augen. u. Ohrenh. (pt. 2) **3**:1-34, 1874.

fever when 14 years of age; this disease had left her with otorrhea, which had persisted until a year before. The growth of the tumor was very slow. (This case was also mentioned by Gruber in his text "Diseases of the Ear.")

Korösi [18] reported the case of a patient 30 years old from whom he had removed a wedge-shaped osteophyte which was pointed downward and attached at the digastric sulcus with a broad base. There was no pathologic report, but roentgen examination showed it to be of uniform density, attached to the medial surface of the mastoid and extending as far as the stylomastoid foramen. There was some question whether this was the result of myositis ossificans.

Korösi [18] reported a second case, that of a 17 year old girl suffering from bilateral suppuration of both ears since childhood, with an exostosis of the mastoid the size of a pigeon's egg.

Waggett and Davis [19] reported the case of a woman 36 years old with a complaint of deafness of four months' standing and a tumor the size of a bantam's egg in the region of the mastoid process, obstructing the external auditory meatus. The contour of the tumor was sharply defined, the mass being demarcated from the outer table by a groove. The tumor was removed with an electric burr and a few taps of a chisel. It was ovoid, consisting of dense bone 1 by 1 by 5/8 inch (2.5 by 2.5 by 1.5 cm.) in size. An uneventful recovery was made.

Bruzzi [20] reported the case of a woman 21 years old with a history of pain and uneasiness in the right mastoid. During the latter part of this time there was a hard projection in this region. The growth increased in size slowly and was the size of a large nut, with a definite outline. The ear was normal. The growth was removed with a gouge, with laying open of the dura mater of the posterior cranial fossa and the first portion of the sigmoid sinus. An uneventful recovery was made.

Politzer [21] reported 2 cases. In the first case there was an osteoma half the size of a large walnut and in the second case an osteoma the size of an entire walnut. The growth in the second case was sharply defined, extending above the level of the mastoid process and by bulging of the posterior wall of the meatus causing occlusion of the lumen of the external auditory canal.

Weinlechner [22] reported the case of a woman with an osteoma of the mastoid process the size of a hen's egg, which was removed. Removal was followed by suppuration of the middle ear, which soon returned to normal.

Fränkel [23] reported the case of a man 58 years old who came for examination with a hard growth on the right mastoid process, which had been present a long time. This growth was about the size of a pigeon's egg; it was cone shaped,

18. Korösi, A.: Ein Fall von Osteoma am Warzenfortsatz, Monatschr. f. Ohrenh. **42**:1501-1502, 1928.

19. Waggett, E. B., and Davis, E. D.: Osteoma of the Mastoid, Proc. Roy. Soc. Med. (Sect. Otol.) **6**:60, 1912-1913.

20. Bruzzi, B.: Osteoma dell'apofisi mastoide, Boll. d. mal. d. orecchio, d. gola, d. naso **51**:263-266, 1933.

21. Politzer, A.: Text-Book of Diseases of the Ear, translated and edited by M. J. Ballin and C. L. Haller, ed. 4, London, Baillière, Tindall & Cox, 1902, p. 636.

22. Weinlechner, J.: Osteom in der Warzengegend durch Operation entfernt; Eröffnung der Warzenzellen; consecutive eitrige Mittelohrentzündung mit Durchlöcherung des Trommelfelles; Heilung, Monatschr. f. Ohrenh. **20**:341-343, 1886.

23. Fränkel, I.: Osteom des Processusmastoideus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:623-626, 1932.

and its lower edge began at the mastoid process at about the middle of the external ear and extended upward into the squamous portion of the temporal bone. The roentgenogram showed a neoplasm of compact bone. The patient allowed removal of only enough of the tumor to give a good cosmetic result.

Florency [24] reported the case of a woman 25 years of age suffering from an exostosis of the mastoid process, which caused excruciating pain but was not sensitive to pressure. The tumor was the size of a hazelnut and was removed with a curet. The pain disappeared, and there was complete recovery, with no recurrence.

Haymann [25] reported the case of a man 24 years old, with a history of a hard, painless tumor of gradually increasing size and of six years' standing on the right mastoid process. There was no history of trauma. The tumor was the size of walnut, pushing the auricle somewhat forward. The lumen of the canal was normal, and the hearing was normal.

An operation was performed with the region under local anesthesia. The periosteum was closely adherent and was removed from the tumor with difficulty. The tumor was hemispheric; it was located over the middle fossa, especially the squamomastoid suture, extending to the upper end of this suture in the region of the parietal incisure. The tumor seemed to ooze out of the portion of the suture. It was removed with a hammer and a chisel. It weighed 9.7 Gm. and measured 3.4 by 2.2 cm. Its surface showed many small nodules. It was a typical osteoma eburneum.

Bourjois [26] reported the case of a woman 40 years old with a hard tumor the size of a pigeon's egg at the end of the mastoid process. The growth had been noticed two years previously. Operation was refused. Later the patient came for treatment on account of parietal pain. A roentgenogram showed the tumor to be an osteoma.

An operation was performed, with great care on account of the close proximity of the meninges and the lateral sinus. The tumor was detached in toto. The lateral sinus was partially exposed. The tumor seemed to have emanated from the sigmoid sulcus.

The pathologic diagnosis was eburnated osteoma.

Kretschmann [27] reported 2 cases of osteoma of the cranial bone. His first case was that of a girl 20 years old who had a tumor on the right side, at the base of the mastoid process. There was a history of chronic suppuration of the right ear. The growth had developed gradually during the past five years. It was hemispheric, hard and firmly adherent to the bone, with a base 2.2 cm. in width and 2.5 cm. in height. It was removed at its base with a straight chisel. During the next six years there was no recurrence.

His second case was that of a girl 19 years of age with a tumor of the right mastoid, present for fifteen years. There was no history of pain, and the tumor had increased in size gradually. The ear was normal. The tumor had a broad

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24. Florency, R.: Exostose de l'apophyse mastoïde; opération; guérison, Paris méd. **11**:157, 1886; abstracted, Monatschr. f. Ohrenh. **21**:17, 1887.

25. Haymann, D.: Zur Kenntnis der Knochengeschwülste des Warzenfortsatzes (Osteoma eburneum processus mastoidei), Ztschr. f. Ohrenh. **77**:23-32, 1919.

26. Bourjois, H.: Ostéome de la mastoïde, Arch. internat. de laryng. **5**:487-488, 1926; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **60**:403-404, 1927.

27. Kretschmann: Osteome an Schädelknochen, Verhandl. d. deutsch. otol. Gesellsch. **19**:304-313, 1910.

base (the diameter was 0.6 cm.). It was removed for cosmetic reasons. The periosteum was firmly adherent to the tumor. The base was severed with a few blows of the chisel. The tumor was spherical for the most part but was attached to an irregularly shaped piece of bone of the external table.

The specimen was the size of a walnut and had fibrous tissue on its outer surface, somewhat elastic to pressure. On section there was a distinct fibrous capsule from which numerous septums found their way into the interior of the tumor, forming a system of cavities in which lay ovoid hard formations ranging in size from that of a pinhead to that of a rice grain. These formations were easily removed from their pedicles and showed a shiny surface.

Gruber,[28] in discussing osteomas of the mastoid region, mentioned three specimens in his collection. One of these was a specimen presented to him by Zuckerkandl. It consisted of two small osteomas, one from over the region of the mastoid antrum and an even smaller one from the squamous portion. The second specimen was a spiniform osteoma about 1 cm. long on carious bone above the external auditory meatus. The third specimen was a rather large osteoma operated on by Weinlechner. This case is reported elsewhere in this paper.

Ventura-Gregorini [29] reported the case of a woman 44 years of age who had had a tumor of the mastoid for four years. It was removed for cosmetic reasons. It was entirely limited to the cortex and was about the size of a cherry.

Stuart [30] reported the case of a woman aged 26 years who had complained of severe headaches and a painful, tender mass behind the left ear, which had been present only a few months. The hearing was not affected. Roentgen examination showed a dense shadow lying lateral to the tip of the mastoid. The tumor was removed in one piece with the chisel and was reported by the pathologist as exostosis eburnea.

There follow abstracts of the 9 cases of the slightly movable type of osteoma.

Smith[31] reported the case of a boy 16 years old who had a history of rapidly increasing deafness in his left ear. On examination there was a mass in the external auditory meatus, on which traction was made, producing vertigo and some bleeding. The next day the patient was seen, and a large, fibrous polypoid growth was observed, which filled the entire left canal. After this growth was removed an extensive hyperostosis of the posterior wall of the external auditory meatus was seen, which prevented a view of the drum. There was a yellow discharge with a foul odor. It was then decided to do a radical mastoidectomy.

The mastoid was of the sclerotic type, and the antrum and tympanic cavity were extensively involved in the necrotic process. The tumor was located in the inferior and posterior part of the antrum and filled the concavity caused by the drooping of the canal adjacent to the hyperostosis.

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28. Gruber, J.: A Text-Book of Diseases of the Ear, translated by E. Law and C. Jewell, ed. 2, London, H. K. Lewis, 1893, p. 322.

29. Ventura-Gregorini, F.: Sopra un raro case di neoformazione ossea della mastoide, Arch. ital. di otol. **49**:379-385, 1937.

30. Stuart, E. A.: Osteoma of the Mastoid, Arch. Otolaryng. **31**:845-847 (May) 1940.

31. Smith, S. M.: An Unusual Growth of the Mastoid Process: Fibro-Chondro-Osteoma of the Mastoid Antrum, Ann. Otol., Rhin. & Laryng. **15**:218-220, 1906.

The tumor was an ovoid mass 1 cm. long, 6 cm. wide and 3 cm. thick and weighed 2 Gm. Its surface was smooth, glossy, slightly nodular and grayish pink. The tissue was hard at some points and soft and elastic at others.

The microscopic report stated that the sections were almost completely covered by stratified squamous epithelium. At some points the four layers of epidermis were to be made out, while at other places but a few rows of stratified flat epithelium were present. That part of the tumor not covered by skin was formed by a band of fibrous tissue which was probably a point of attachment of the specimen. Beneath the skin were areas composed of loosely woven strands of fibrous tissue transmitting numerous small blood vessels. In some areas the perivascular structures were densely infiltrated with mononuclear leukocytes. The sections also contained trabeculae of fibrocartilage and here and there denser (osteoid) areas. There were also many large lymph sinuses.

The diagnosis was fibrosteochondroma of the mastiod.

Borrás [32] reported the case of a boy 11 years old who had a hard, almost immobile mass the size of a hazelnut in the right mastoid region, present for two years. The base of implantation of the tumor was 2 cm. in diameter.

With the boy under ether anesthesia and with a perpendicular incision the tumor was removed from its insertion with a hammer and a chisel. Between the slightly flattened surface of the tumor and the external surface of the mastoid which was its seat, there was a fibrocartilage which united the two bones, forming a pseudarthrosis.

Green [33] reported 2 cases of osteoma of the mastoid. The first case was that of a boy 16 years old who had been somewhat deaf for several years without earache or suppuration at any time. Three weeks before he consulted Green there were redness, swelling and pain just below the ear, and a superficial abscess was seen between the mastoid and the jaw, which was opened. The external auditory meatus was filled with a hard, smooth growth rising from the posterior wall.

In a few days there was an increase of pain and discharge from the meatus, coming from behind the growth. Soon the tissues over the mastoid became red, swollen and edematous and there was slight movement of the mass in the meatus.

With the patient under ether anesthesia an incision was made as for a mastoid operation and a movable, hard mass on the anterior surface of the mastoid was exposed and easily removed. Its removal cleared and exposed the entire meatus. The growth lay embedded in the mastoid and was surrounded by caseous material, epidermal flakes and a small amount of pus; the bony walls of the cavity were carious superficially.

The tumor was of irregular shape and measured 19 by 15 by 9 mm. It consisted of true bone in its interior, showing well developed bone corpuscles, and the outside was entirely covered with a layer of cartilage which had ulcerated in two spots where the underlying bone was carious superficially.

The second case was that of a man 21 years of age, with a history of deafness in the right ear for several years. He had had no pain or discharge for the past ten weeks. Examination showed the right meatus completely closed by a hard bony tumor which was slightly movable. There was a slight purulent discharge.

With the patient under ether anesthesia, through the usual mastoid incision, the tumor, which constituted the posterior wall of the external wall of the external

32. Borrás: Osteoma del región mastoidea, Bol. ofic. d. col. de méd. de Barcelona **2**:123-127, 1898.

33. Green, J. O.: Osteoma of the Mastoid, Arch. Otol. **22**:280-283, 1893.

auditory wall of the meatus, was found freely movable and easily removed with forceps. The bony cavity contained detritus, and the walls were carious superficially. The growth was 12 mm. long and 12 mm. thick and was covered with a layer of fibrous tissue. The center was of true bone with well formed bone corpuscles.

Leoni [34] reported the case of a youth 19 years of age with a hard nodule in the region of the left mastoid, which had gradually increased in size for the past six months. The nodule was the size of a small nut; the ear was normal.

Roentgen examination showed a tumor mass adhering to the mastoid by a large pedicle at the level of the base. The structure of the formation was characterized by islets of eburnated substance separated from each other by less compact bone. With a few strokes of a gouge the pedicle, which corresponded to the upper half of the mass, was removed; the lower half of the tumor was in contact with the mastoid, with interposition of a fold of periosteum. All the periosteum which covered the tumor was thicker than that which covered the mastoid.

The tumor measured 21 by 21 by 8 mm.

Bareischnikoff [35] reported the case of a man 23 years of age with a tumor in the left temporal region. The growth had been present practically all his life and had enlarged very little. It was at the level of the zygomatic process, 2 cm. from the junction of the auricle with the head. The enlargement was mobile, not adherent to the skin and the size of a large cherry; it protruded outward from the skull. The tumor was removed through an incision in the skin and was found to be in the subcutaneous tissue between the skin and the zygomatic process. Its dimensions were 1.6 by 1.4 by 1 cm. and it weighed 1.8 Gm.

Bareischnikoff said that this is of particular interest because many writers, especially Petroff, have denied the existence of osseous osteomas in the soft tissue, saying that osteomas grow because of their own elements and not because of hyperplasia of the surrounding tissues.

Coates [36] reported the case of a man 39 years of age who had a small osteoma at the superior posterior angle of the left mastoid. This osteoma was 1½ inches (3.7 cm.) in diameter and ½ inch (1.2 cm.) in thickness; it was hard but not ivory-like. It was removed with little difficulty with a flat chisel, as it was attached to the cortex only.

Friedberg [37] reported the case of a Negro 52 years of age with a slowly growing osteoma of the right mastoid which had been present ten years. Functional tests of the cochlea and of the vestibule revealed no abnormality. Roentgen examination showed a well outlined mass of the right mastoid.

The tumor was covered by a layer of periosteum, from which it was easily separated. The mass was firmly attached to the cortex of the mastoid and was easily removed with a gouge without uncovering the dura or the lateral sinus. The specimen was hemispheric and measured 3 by 2.5 by 1.5 cm.

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34. Leoni, P.: Osservazioni sopra un caso di osteoma della mastoide, Boll. d. mal. d. orecchio, d. gola, d. naso **52**:490-495, 1934.

35. Bareischnikoff, J.: Sur les anomalies de genèse de l'os temporal, Ann. d'oto-laryng., July 1934, pp. 708-709.

36. Coates, G. M.: Osteoma Growing from the Mastoid Cortex, Arch. Otolaryng. **28**:27-28 (July) 1938.

37. Friedberg, S. A.: Osteoma of the Mastoid Process, Arch. Otolaryng. **28**:20-26 (July) 1938.

Hempstead [38] reported the case of a woman 22 years of age with a large osteoma of the left mastoid of more than a year's standing. The middle ear was not affected, and the hearing was normal. The osteoma, which was attached to the tip of the mastoid, was removed with a chisel with the patient under general anesthesia. The growth measured 6.5 by 5 by 3.5 cm.

The third group, consisting of Amberg's case and my own second case, is probably the most interesting and certainly the most unusual of these groups.

Amberg [39] reported the case of a woman 41 years old with a history of suppuration of the ear for a few days. There were pain in the mastoid and chills. The mastoid was opened, and no pus was encountered; the tip was evacuated, and the bone was soft. The patient died of abscess of the brain and meningitis. Amberg reported this case because of the peculiar finding in the diploic mastoid region. The outer surface of the mastoid did not present anything unusual. In the mastoid, however, he found a pearl-like bone formation about double as large as the head of the hammer, which was apparently attached with a small stem and showed a minute sharp-pointed process. This specimen measured 3.5 by 3 by 2.5 mm. The attachment was somewhat curved, 3 mm. long and from 0.5 to 0.1 mm. thick. Amberg could not tell where it originated, but he found it somewhat removed from the cortex in the mastoid cavity. He stated that it is not impossible that it was dislocated by the chisel from the inner wall of the cortex, but mentioned this as a remote possibility.

REPORT OF PERSONALLY OBSERVED CASES

CASE 1.—A woman 28 years of age had what seemed to be a bony growth back of her left ear, which had been present fourteen years and had gradually increased in size. There had been a bilateral suppuration of the ears for twenty years.

Examination showed a large central perforation of both ears, with a mucopurulent discharge. The hearing was bilaterally limited to a whisper at the ear. There was a mass protruding from the central portion of the mastoid process; it was hemispheric, smooth and painless. The base was circular and about 1 inch (2.5 cm.) in diameter, protruding above the surrounding area about ¾ inch (1.9 cm.). Roentgen examination showed a well localized area of increased density of the mastoid, and a diagnosis of osteoma of the mastoid was made.

With the patient under ether anesthesia a vertical incision was made over the protruding mass. The skin and soft parts, including the periosteum, were easily detached and retracted from the surface of the growth. The tumor had a wide base. During its removal the dura was exposed superior to the antrum. After removal of a considerable amount of bone with a chisel at the base of the tumor, the mass was broken away with a mallet, a small exposure of the lateral sinus being left. Otherwise a thin layer of bone was left over the posterior and middle fossae. The bone which was left bled rather freely and did not seem to be

38. Hempstead, B.: Osteomas of Paranasal Sinuses and the Mastoid Process, J. A. M. A. **111**:1273-1276 (Oct. 1) 1938.

39. Amberg, E.: An Irregularly Pearl-Shaped Bone Formation in the Mastoid Process, New York M. J. **92**:709, 1910.

Fig. 1 (case 1).—Appearance of the osteoma before operation.

Fig. 2 (case 1).—Roentgenogram of the mastoid region.

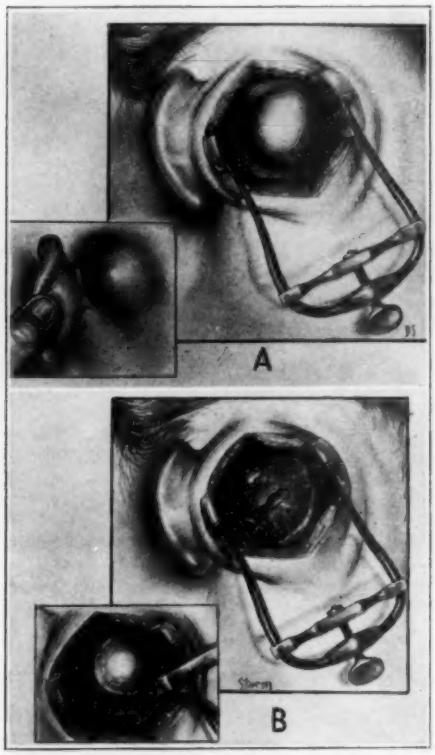

Fig. 3 (case 1).—*A*, the insert is a sketch of the mastoid region, showing the osteoma covered with skin. The main drawing shows the tumor as it appeared after the soft parts were incised and removed. *B*, the insert is a sketch of the osteoma being removed with a chisel. The main drawing shows the wound after the tumor has been removed. Three mastoid cells are seen in the lower portion of the drawing. The lateral sinus is partially uncovered in the middle portion, while the dura is shown exposed in the upper portion.

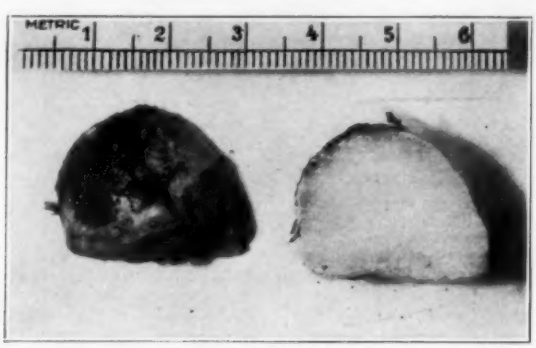

Fig. 4 (case 1).—Photograph of the removed osteoma.

extraordinarily hard. Some further removal of the bone at the base of the mass was done with chisels and curets. After removal of the tumor mass the semiradical operation was completed.

The pathologic report was as follows:

Macroscopic Picture.—The specimen consisted of a hemispheric bony mass measuring 2.7 by 2.2 by 2.5 cm. and weighing 14 Gm. It was attached by a broad

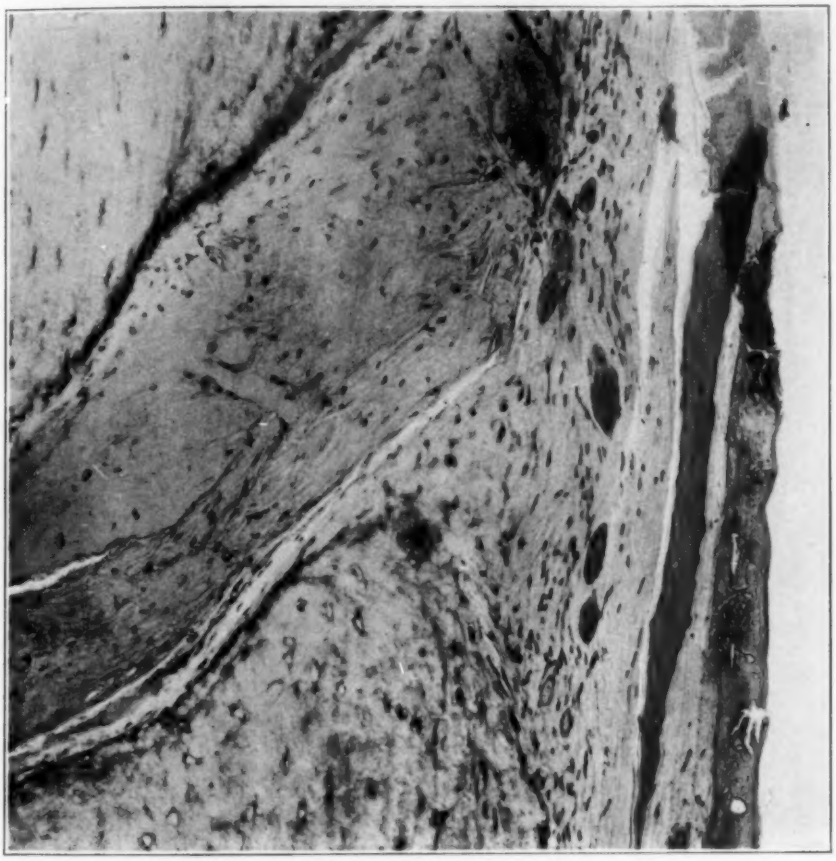

Fig. 5 (case 1).—Photomicrograph of a compact osteoma of the mastoid process. The vascular periosteum sends blood vessels and fibrous tissue by means of Volkmann's canal into the periosteal lamellae, between which are lacunas with communicating canaliculi containing branching bone corpuscles. × 200.

base and partly covered by a delicate pink membrane. The sectioned surface was light gray firm bone, slightly darker about the periphery. The membrane previously mentioned was seen on section (fig. 3 *B*).

Microscopic Picture.—The structure was similar to that of membrane bone. Volkmann's canals pierced the periosteal lamellae carrying blood vessels surrounded by fibrous tissue from the periosteum into the compact bone. The peri-

osteal lamellae, with the lacuna, canaliculi and bone corpuscles, had been laid down parallel to the periosteum. The haversian canals contained fibrous tissue, small arterial and large venous capillaries, nerves and lymphatics. The lacunas communicated with each other by means of similar canaliculi. Each lacuna was occupied by a flattened, nucleated cell which seemed to send branches along the canaliculi.

The diagnosis was compact osteoma of the mastoid process.

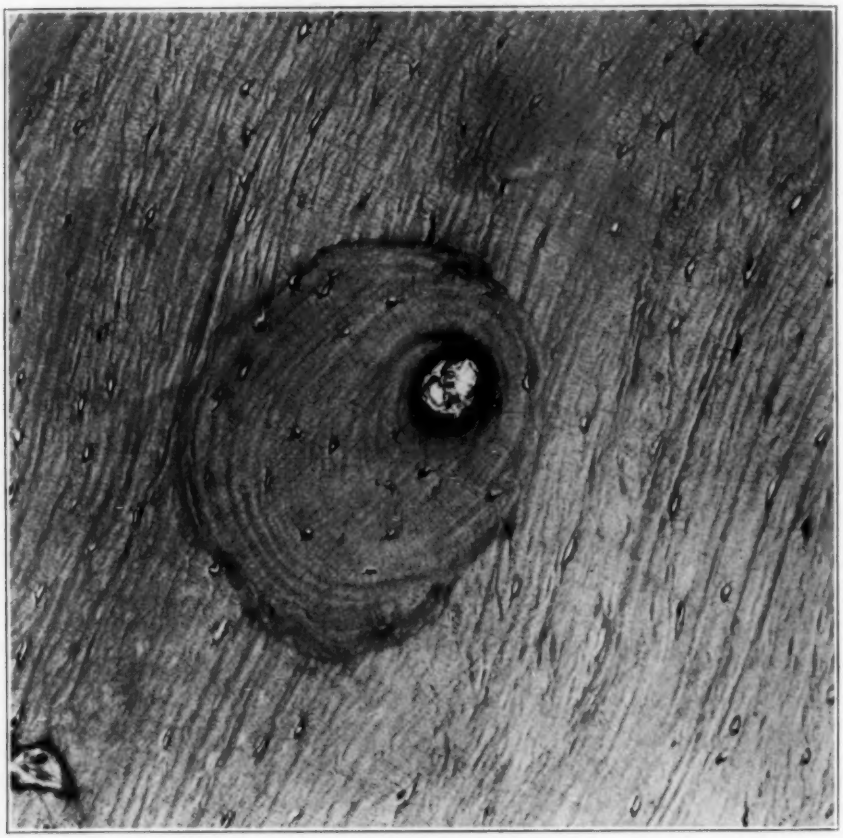

Fig. 6 (case 1).—Another photomicrograph of the osteoma. Note the haversian canal with small arterial and large venous capillaries; the concentric lamellae of fibrous tissue, and the lacunas with communicating canaliculi, containing bone corpuscles. × 300.

It seems to me that the growth in this case was rather typical of the most common type of osteoma of the mastoid. It did not obstruct the external auditory meatus; there was chronic suppuration of the ear; it was of slow growth and without pain; in its removal the dura and the lateral sinus were exposed, and an uneventful recovery was

made. All these features classify it under the most usual type of osteoma of the mastoid (type I).

CASE 2.—A woman 60 years old had had suppurative otitis media for about six weeks. The discharge had had a bad odor for the few weeks just before she

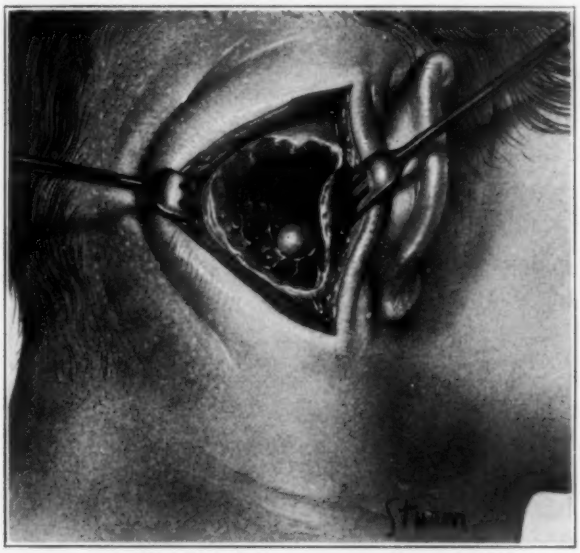

Fig. 7 (case 2).—Sketch of the growth as seen during the operation.

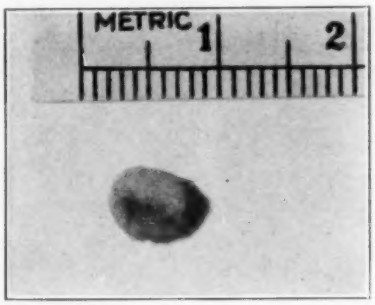

Fig. 8 (case 2).—Photograph of an osteoma of the right mastoid.

came to my office, and in that time pain had developed in her right ear and in the right side of her head.

Examination showed a central perforation of the right membrana tympani, with a badly swollen mucous membrane protruding through the perforation and a profuse purulent discharge with a bad odor. Hearing in the right ear was limited to a whisper at 6 inches. The left ear was normal. A roentgenogram

showed a pneumatic type of mastoid on both sides, with increased density of all cells on the right. Nothing unusual was noted.

The following day a semiradical mastoidectomy was done on the right side. There was extensive cell formation, and all the cells were filled with dark straw-colored fluid and swollen mucous membrane, some of which was granular. The bone of the entire mastoid was very soft, and many septums were broken down. The seventh nerve was exposed in its bed just below the knee for about ¾ inch

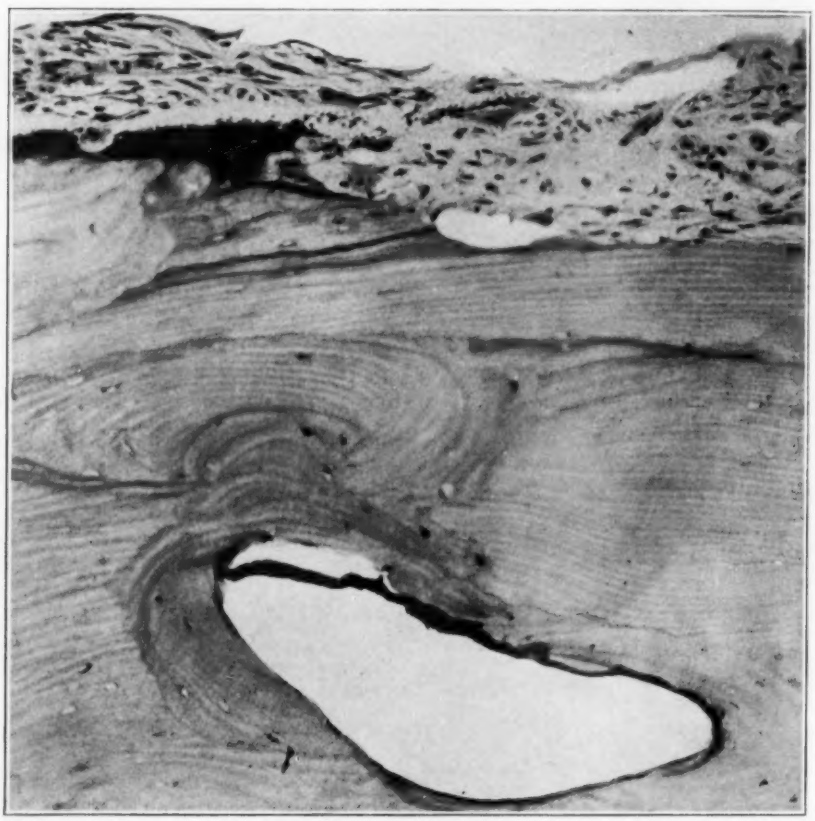

Fig. 9 (case 2).—Photomicrograph of an osteoma of the right mastoid. The bone shows lamellae, lacunas, canaliculi, corpuscles, a small area of calcium, a marrow space lined by endosteum and the periosteal covering. × 200.

(1.9 cm.). Well toward the tip of the mastoid, in a large cell, was a loose, hard, ivory-like mass, seemingly unattached. It was slightly roughened on one side; otherwise it was perfectly smooth.

The pathologic report was as follows.

Macroscopic Picture.—The specimen consisted of a grayish white, opaque, firm globoid mass 0.5 by 0.5 by 0.5 cm. Except over a small area its surface was smooth.

Microscopic Picture.—The specimen was a section of decalcified bone with lacunas and canaliculi. In the lacunas were bone corpuscles. The outer surface of the bone was covered by periosteum, and the bone contained small and large marrow spaces lined by endosteum. These were mostly empty.

The diagnosis was osteoma.

COMMENT

In résumé, I should like to give some of the more important observations in all of these cases, including my own.

Of the entire 30 cases, there was suppurative otitis media of the affected ear in 10, and there was suppuration of the middle ear as a result of operative removal of the mastoid in 1.

Suppuration of the mastoid has been given as an etiologic factor in formation of osteomas of the mastoid, but it seems to me that there is probably no connection. In the 17 cases of osteoma of the mastoid in which there was no suppuration, the tumor had practically the same appearance macroscopically and microscopically as those with which suppuration occurred and the clinical progress and the final results were the same in the two types of case. No complications seem to have arisen at any time on account of suppurative mastoiditis coincident with removal of the osteoma, but certainly suppuration of the mastoid cells surrounding the growth makes the operative procedure much more formidable. This is especially so when the dura and the lateral sinus are uncovered during the operation. Certainly if there is a tear in the dura or in the lateral sinus and suppurative mastoiditis is present, the danger of meningitis or infection of the blood stream is greatly enhanced.

In 5 cases there was obstruction of the meatus due to proximity of the tumor to the canal, and the hearing was affected in 1 case only by complete closure of the canal by the osteoma. These conditions were relieved by the removal of the growth.

It seems to me rather remarkable that the seventh nerve was not affected by the growth itself in any of these cases and still more remarkable that the function of the nerve was at no time disturbed during operative removal of these growths. In my second case the seventh nerve was exposed for a distance of ¾ inch (1.9 cm.), owing to the suppurative condition of the mastoid rather than to the position of the tumor.

In the entire group of 30 cases there was only 1 in which there was disturbance of the internal ear; this was the case of Paulian and Popp,[14] in which a small exostosis compressed the superior semicircular canal, causing a hemorrhage of the labyrinth on the right side and producing both vertigo and deafness.

There was only 1 case in which the tumor was described as definitely pedunculated—the case reported by Leoni.[34] In such a case removal is

a minor procedure as compared with removal of an osteoma with a wide base and especially an osteoma with attachment to the inner table.

In a study of the technic of removal one observes that opening of the mastoid cells was mentioned four times. It seems to me that extensive pneumatization in the case of an osteoma of this region greatly facilitates operation.

In my first case there were only a few mastoid cells; they were small, and the bone was of the sclerotic type. The osteoma had a very wide base and was removed with considerable difficulty, much more so than if there had been extensive cell formation.

Completely detached osteomas, like that in Amberg's case and that in my second case, is entirely different from the usual osteoma of the mastoid. The osteoma in my own case was lying loose in a large mastoid cell, the picture being similar to that described by Amberg.

In the entire group of cases the dura was exposed four times and the lateral sinus four times as the result of operative intervention. Both the lateral sinus and the dura were exposed in the cases of Preusse [16] and Bruzzi [20] and in my own first case, while the dura alone was exposed in Burger's [15] case and the lateral sinus alone in Bourjois' [26] case.

No complication resulted from any operative procedure, even though the dura was exposed with accompanying suppurative mastoiditis in Burger's case and there was exposure of the dura and the lateral sinus with suppurative mastoiditis in my own first case.

CONCLUSIONS

The following conclusions were reached as a result of the study of these cases:

1. Complications resulting from the presence of an osteoma or from operative removal of an osteoma practically never arise.

2. The growth rarely, if ever, recurs.

3. There is considerable evidence to indicate that the origin of the usual osteoma of the mastoid is from preosseous connective tissue rather than from remnants of cartilage.

4. The final result of treatment is usually satisfactory.

The pathologic reports of specimens in these cases were made by Dr. Harry C. Schmeisser, director of the Pathological Institute, University of Tennessee. The illustrations are the contribution of Dr. Joseph L. Scianni, artist of the Pathological Institute, and of Miss Dorothy Sturm.

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ABSTRACT OF DISCUSSION

DR. GEORGE M. COATES, Philadelphia: When I came across my case of osteoma of the mastoid, reported in 1938, although it was the only one I have observed, I was not aware of the comparative rarity of this condition, at least as shown by the published reports, and I was therefore interested in the case of Dr. Stanton M. Friedberg, who reported his case at the same time. I am grateful to Dr. Simpson for this exhaustive and comprehensive paper, which brings up to date an interesting subject.

In most discussions of osteoma of the mastoid reference is made to hyperostoses or exostoses of the external auditory canal, and some confusion seems to exist in regard to their relationship. From several of the reports cited or abstracted by Dr. Simpson, I get the impression that some authors have used these terms interchangeably. While these processes are undoubtedly similar, if one accepts the fact that all exostoses of the external auditory canal can be classed as osteomas the field will be immensely widened, for the former are very common, not only in the old but at times in the young. When there is a distinctly pedicled bony growth in the external canal, it may be an osteoma, a true neoplasm, but a general bony thickening, even though lumpy, is probably no more than an osteogenetic process, such as is seen frequently in the nasal septum or at times in the frontal sinus.

True osteomas of the mastoid display the same characteristics as osteomas in other locations. They may be sessile, pedunculated, compact, eburnated or cancellated. Suppuration in the neighboring middle ear or ethmoid cells does not seem to be concerned in their production. Their cause is definitely unknown. They may involve the cortex only or may arise from or extend into the mastoid cells and be incorporated in the internal table. They are often symptomless, but they may cause pain or, as in 1 case abstracted in this report, symptoms by pressure on contiguous structures. Except in such cases these growths are benign and harmless, but they may cause external deformity, as in my patient; the lump behind his ear embarrassed him and annoyed him when he was brushing his hair. Some of these growths, those having pedicles, are easily removed when once exposed. Those on the cortex are also not difficult to remove. They are covered with periosteum which is removed without too great difficulty, although it may be somewhat more adherent than that covering normal cortex. In my case the rather large growth was pried loose from its bed with a broad gage, leaving a somewhat shallow depression without exposure of any cells. When the internal table is involved in the growths, their removal necessitates resection of this plate with exposure of the dura. This is a common occurrence in operations on the mastoid for suppuration and should, therefore, cause no difficulty or complications even in those few cases in which there is a concomitant infection of the mastoid. These neoplasms are seldom dangerous but, on account of their apparent rarity, are interesting curiosities. When encountered they should be reported.

PLUMMER-VINSON SYNDROME

REPORT OF TWO CASES

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The Plummer-Vinson syndrome was described by Vinson [1] in his recent book on diseases of the esophagus under the title of "hysterical dysphagia." According to him, a primary inability to swallow solid food may be followed by a train of secondary symptoms of more or less severity—anemia, glossitis, splenomegaly and achlorhydria. The dysphagia has a neurotic basis, a fear of choking. There is no organic lesion of the esophagus. A review of the rather voluminous literature shows that in this opinion he is almost alone.

In a recent paper on this syndrome, Cordray [2] stated that web formation at the mouth of the esophagus is the cause of the dysphagia. In his brief review of the literature he cited a number of authors as having found various lesions of the mouth of the esophagus which were supposed to have been the important factor in the dysphagia. He did not account for the appearance of the webs, stating only that severe experimental anemia will not cause them in rats. Here are two opinions, then, so far apart that one wonders if the same disease can be in question. One bases the whole trouble on a neurosis without an organic lesion. The other considers that organic lesions are always present. Once the dysphagia is established, by whatever cause, the other symptoms mentioned by both authors are caused by partial starvation.

In reading the many papers which have been written on this syndrome one gains the impression that the disease presents itself in many forms, now this and now that symptom being prominent. Each author naturally emphasizes the aspect which has come most strongly to his attention. Thus, by some the anemia is carefully described and the dysphagia scarcely mentioned. Others deal with certain gastric symptoms, such as achlorhydria with accompanying anemia, and make little mention of oral and pharyngeal manifestations. Still others consider that the dysphagia is primary, all the other symptoms being secondary

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1. Vinson, P. P.: The Diagnosis and Treatment of Diseases of the Esophagus, Springfield, Ill., Charles C. Thomas, Publisher, 1939.

2. Cordray, D. P.: Ann. Otol., Rhin. & Laryng. **49**:160, 1940.

developments. One must conclude that the syndrome scarcely ever presents itself as a whole. Only one feature may be developed, and that slightly.

As is the case with many other human ailments named for authors, those for whom this syndrome was named were late on the scene with their description. In a dissertation on "dysphagia hysteria," published in 1893, Blankenstein [3] gave an accurate description of the Plummer-Vinson syndrome. He stated that nervous persons, as well as anemic and chlorotic ones, are predisposed to spastic strictures of the esophagus, which are relieved by the passage of sounds. Then he reported that in hysterical persons dysphagia occurs owing to lack of desire for deglutition. A number of such persons on esophageal examination showed no abnormality, in the esophagus, the pharynx or the mouth. His treatment consisted of passage of sounds, reassurance of the patient, and passage of a constant electrical current through the neck at meal times. This brought about great improvement. In his report, it will be seen, only the dysphagia was emphasized.

In 1909 and 1913, Faber [4] published papers on "achylia gastrica with anemia," based on 300 cases. He did not emphasize dysphagia. The combination of gastric symptoms and anemia appears in many later papers.

In 1913, Bronner [5] published some notes on painful lesions at the mouth of the esophagus. He compared the mouth of the esophagus to the cardiac orifice of the stomach and stated that spasm is equally possible at either. He noted that these lesions occurred most frequently in women from 30 to 50 years of age, who often also had dry catarrh of the pharynx. He reported 2 cases, that of a woman of 30 and that of a woman of 55. Both had painful fissures in the mouth of the esophagus, which healed under treatment. This author expressed the opinion that nonmalignant ulcers and fissures at the mouth of the esophagus are not uncommon and that they are connected in someway with dry catarrh of the pharynx.

The syndrome appeared in the American literature in 1911. Clark [6] in that year described at a meeting of the American Laryngological Association a case of partial web in the upper part of the esophagus. In the same year, Mosher [7] published a short paper on "Folds and Webs at the Upper End of the Esophagus." He made the significant remark that such webs are so easily ruptured by the passage of sounds, and any obstruction thus relieved, that one is tempted to consider the

3. Blankenstein, P.: Dysphagia hysterica, Thesis, Bonn, J. Bach Wwe., 1893.

4. Faber, K.: Berl. klin. Wchnschr. **50**:958, 1913; Med. Klin. **5**:1310, 1909.

5. Bronner: J. Laryng., Rhin. & Otol. **28**:32, 1913.

6. Clark, J. P.: Tr. Am. Laryng. A., 1911, p. 187.

7. Mosher, H. P.: Laryngoscope **27**:235, 1927.

condition neurasthenic, whereas the obstruction is actually a physical narrowing of the passage.

In 1912, Plummer [8] mentioned spasm at the upper end of the esophagus and discussed the differential diagnosis of the various causes of dysphagia. In 1922, Vinson published a report based on 69 cases. He considered the cause of the dysphagia to be a hysterical spasm. He found no organic lesion of the esophagus and stated the opinion that the anemia and glossitis are due to the dietary deficiency caused by the dysphagia.

The first important paper on this subject in the English literature was that written by Kelly [9] in 1919, entitled "Spasm at the Entrance of the Esophagus." His paper gives such a complete description of the syndrome that it will be summarized at some length. The patients were women between 40 and 50 years, whose complaint was dysphagia of several years' duration. Some had a history of anemia, dyspepsia or impaired general health, and several were neurotic. The dysphagia was referred to the level of the larynx and resulted in extreme nervousness in regard to eating, especially in the presence of strangers.

Examination of the mouth and pharynx usually revealed nothing, though in some cases the mucosa had a pale, waxy aspect and the tongue was smooth and devoid of papillae. Fissures at the angles of the mouth were also noted, but all these appearances were regarded as the consequences of the dysphagia. In 1 case inspection revealed a circular web reducing the lumen of the esophagus. Rupture of this web gave complete relief. The question was therefore asked: Is the obstruction due to a spasm, or to an organic stricture resulting possibly from cicatricial contraction?

Ten cases were studied in which the dysphagia had been present from two to thirty-three years. The deepest part of the hypopharynx did not present the usual sphincter-like appearance. Tense bands passed in various directions. The entrance to the esophagus was a pinhole-like opening, not always in the midline. Sometimes half the mouth of the gullet seemed closed by a web passing backward from the cricoid cartilage.

In spite of these appearances, it was found not difficult to pass sounds, the sound often slipping in suddenly as if a spasm had yielded. The results of this treatment were excellent. The author concluded that, in spite of the presence of webs, the cause of the dysphagia was usually spasm and only exceptionally membranous stricture. He excluded hysteria, however, as the cause of the spasm, as in cases of hysterical dysphagia symptoms are intermittent; there is no desire to swallow, and his patients were not neurotic.

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8. Plummer, H. S.: Diffuse Dilatation of the Esophagus Without Anatomic Stenosis, J. A. M. A. **58**:2013 (June 29) 1912.

9. Kelly, A. B.: J. Laryng., Rhin. & Otol. **34**:285, 1919.

All the patients were women. Since in most cases of cancer of the mouth of the esophagus the patients are women, it was suggested that there may be some factor in women predisposing to cancer and spasm at that site.

As to the cause of cicatrices, it was noted that dysphagia frequently set in after impaction of a foreign body, so that injury might be the cause. As for spasm, the author suggested that the balance of the sensory nerve plexus of Meissner and the motor plexus of Auerbach is an important factor in this connection. If the sensory plexus is in a hyperesthetic state or is unduly irritated by a foreign body, a fissure or an inflammatory condition or if the motor plexus is overstimulated, abnormal local contraction or spasm results; spasm once established may tend to be perpetuated as a nerve cell habit.

It might be mentioned here that in a communication to the *British Medical Journal* in 1928 Kelly stated that since Auerbach's plexus does not extend to the upper centimeters of the esophagus it could not influence the action of the sphincter at the entrance. He stated that he still considered the condition a spasm.

At the same time that Kelly's paper was published, there appeared one by Paterson.[10] The description by the latter was almost exactly like that of the former. He noted the thin, stretched appearance of the mucous membrane and warned that any instrument must be passed with great care. He gave the first description of the histologic picture of tissue removed from tongue and cheeks. There were thinning of the epidermal layer and thickening from infiltration of the underlying tunica propria. This might account, it was thought, for the lack of resiliency. He also noted that the supervention of malignant disease at the mouth of the gullet was too frequent to be a coincidence.

His conclusions were as follows: Spasmodic dysphagia in women is sometimes associated with definite changes in the upper food passages. If not the sole cause of the spasm, they increase the tendency. The association produces a more obstinate type of dysphagia.

In a short note, Hurst [11] called attention to the fact that he named the disease the Plummer-Vinson syndrome, as he was then not aware of the earlier work of Kelly and Paterson. The latter authors mentioned splenomegaly and glossitis but gave no details about the anemia.

Hurst reaffirmed his view that the dysphagia is not of hysterical origin (Vinson) nor is it caused by reflex spasm (Kelly and Paterson; Jones and Owen), as Hurst himself at one time was inclined to believe. He held the primary cause to be achalasia of the pharyngoesophageal sphincter of Negus, exactly analogous to achalasia of the cardiac sphincter, which was erroneously described as cardiospasm. Achalasia

10. Paterson, D. R.: Laryng., Rhin. & Otol. **34**:289, 1919.
11. Hurst, A. F.: Brit. M. J. **1**:375, 1928.

of the cardia now is known to be due to inflammatory and degenerative changes in the plexus of Auerbach, preventing normal relaxation of the sphincter when reached by peristaltic waves carrying food down the esophagus. Similar changes may be present in the Auerbach plexus at the upper portion of the esophagus, preventing relaxation at the beginning of the act of deglutition. Such changes could be due to direct spread of inflammation from the mucosa to the deeper-lying plexus. Obviously, said Hurst, atrophic glossitis and pharyngoesophagitis result from streptococcic infection, which is also responsible for anemia and splenomegaly. Relief from dilation of the closed pharyngoesophageal sphincter is comparable to relief obtained with the mercury tube in the presence of achalasia of the cardia. This opinion introduced infection as a cause of the dysphagia of the Plummer-Vinson syndrome.

Having carried the historical review of the Plummer-Vinson syndrome up to the paper by Vinson and found the origin of the name, it will be well to follow the literature less closely and turn to a more general discussion of the separate components of the syndrome, the pathologic picture and other points of interest. These are (1) the anemia, (2) the dysphagia, (3) the glossitis, (4) the achlorhydria, (5) the pathologic picture and (6) the possibilities of carcinoma.

ANEMIA

As has been said, this syndrome has been discussed under many different aspects, according as they presented themselves to each author. Witts,[12] writing in the *British Medical Journal* in 1931, stated that chronic microcytic anemia is much more common in women than in men. It is not due, in his opinion, to any of the common causes of secondary anemia, such as hemorrhage or infection, but may be an independent disease. The anemia is hypochromic, the red cells varying from 2,500,000 to 4,000,000 per cubic millimeter of blood and the value for hemoglobin from 25 per cent to 60 per cent. Achlorhydria precedes the anemia and may remain after the anemia is cured.

The author reported 101 cases, in 69 of which simple achlorhydric anemia was present. Thirteen of the 101 patients had dysphagia as well as anemia. The case of a mother aged 44 was reported who had dysphagia and anemia; her daughter, aged 23, had simple achlorhydric anemia. A hereditary influence is thus suggested.

Glossitis, pharyngitis and dysphagia, if present, are probably due to streptococcic infection of the mucosa of the mouth, spreading from there to the esophagus. The infection destroys the nerve terminals which initiate the swallowing reflex and so causes dysphagia.

12. Witts, L. J.: Brit. M. J. **2**:883, 1931.

Graham and Johnson [13] reported 6 cases of anemia with dysphagia. It was noted that the fragility of the red cells was increased. This observation was considered so notable that the authors suggested that this may be a specific type of anemia associated with slight increase in the fragility of the red cells, with achlorhydria and with secondary changes in the mucous membranes of the tongue, mouth and esophagus and, subsequently, dysphagia at a late stage.

Perhaps the question of anemia in the Plummer-Vinson syndrome may best be covered by summarizing a paper by Thiele and Kühl.[14] The blood picture is characteristic, said these authors; there is a diminished value for hemoglobin in proportion to the diminished erythrocyte count, and the color index is thus always low. The average diameter of the blood cells is below normal. Pathologic erythrocytes are very rare.

They stated the belief that the combination of anemia and dysphagia known as the Plummer-Vinson syndrome is not a specific disease but must be regarded as one special type of essential hypochromic anemia. Owing to the frequency of achlorhydria in connection with the syndrome, one might assume that it is always accompanied by achylia. One might then imagine that it begins as atrophy of the mucous membrane of the stomach, which continues upward to the esophagus. The connection between atrophy of the mucous membrane of the stomach and changes in the upper part of the digestive tract is furthermore suggested by the fact that the syndrome has developed after operations on the stomach. On the other hand, the typical Plummer-Vinson syndrome has been noted without changes in the stomach and without great reduction of the gastric concentration of hydrochloric acid.

These authors also stated that the syndrome occurs only in women, but they have observed it at earlier ages than have most others. The atrophy of the mucous membrane never leads, according to them, to organic stenosis of the esophagus.

Summary.—The blood picture determines essential hypochromic anemia. Occasionally atrophy of the mucous membrane at the mouth of the gullet leads to dysphagia, which confirms the diagnosis, especially if spoon nails and paresthesias are noted. The syndrome is nearly always found in women and is frequently accompanied by achlorhydria and occasionally by a tumor of the spleen.

DYSPHAGIA

As has been said, Vinson considered the dysphagia as developing on a hysterical basis. In the many cases observed and reported by him he found no organic lesion of the esophagus. The anemia and glossitis,

13. Graham, G., and Johnson, R. S.: Quart. J. Med. **18**:41, 1932.
14. Thiele, W., and Kühl, H.: Klin. Wchnschr. **17**:1191, 1938.

which may develop only after years of dysphagia, he considered to be due to prolonged semistarvation. Any changes found in the oral and pharyngeal mucous membrane would also be secondary to the dysphagia and anemia.

The great majority of the other authors writing on the syndrome have considered lesions at the mouth of the esophagus to be the cause of the dysphagia. In some cases, these lesions have taken the form of painful fissures or ulcers, and in other cases they have appeared as webs or bands of fibrous tissue. The mode of operation of these lesions in causing difficulty in swallowing is variously explained. Some say that they actually form a physical barrier which prevents passage of the food. Others are of the opinion that their mere presence interferes with initiation of the swallowing reflex, especially if the changes in the hypopharynx are extensive. The thought is that the atrophy of the mucous membrane may have involved the sensory nerve terminals, so that the presence of food in the hypopharynx does not excite the reflex which causes relaxation of the cricopharyngeal muscle.

Painful fissures and ulcers act somewhat differently. Their presence allows overstimulation of the sensory terminals. The response to this is an actual spasm of the esophageal sphincter, rather than loss of relaxing power.

Summary.—The dysphagia may be caused by (1) spasm due either to hysteria or to the presence of atrophic or painful lesions which lead to understimulation or overstimulation of the sensory arc of the swallowing reflex or (2) by an actual physical barrier formed by bands or webs across the mouth of the esophagus.

How shall one explain the presence of these webs and bands? Certain authors think that a foreign body or some other source of injury creates a lesion which, when healed, leaves a web or a band. The presence of webs has also been explained on the basis of spinal deformity. Those who consider the dysphagia as following the anemia think that the atrophic lesions involving the pharyngeal mucous membrane may first take the form of ulcers, which heal to form scars. Infection of poorly resistant tissue has been suggested as the source of the damage. Finally, one author mentioned the possibility that syphilis might be back of the whole syndrome.

GLOSSITIS

The glossitis takes the form of atrophy of the lingual papillae. The tongue appears smooth and red and is painful. It is shrunken in appearance, not differing much from the tongue in the presence of other severe anemias.

ACHLORHYDRIA

The relation of dyspepsia and the Plummer-Vinson syndrome has been noted by many authors. In fact, many consider the syndrome a phase of one disease, which begins with atrophy of the gastric mucous membrane and goes on to severe anemia and dysphagia. A number of cases have been reported in which the syndrome developed in connection with operations on the stomach. The lesions calling for operative attack were for the most part ulcers of the pylorus. It is thought that lack of hydrochloric acid in the gastric juice interferes with absorption of iron from the food; hence the anemia. It is significant that with the iron treatment recovery is quicker if hydrochloric acid is also given.

Yet it must be said that although patients with the so-called Plummer-Vinson syndrome often show lack of free hydrochloric acid in the gastric contents, many show no such deficiency. Achlorhydric anemia and the syndrome are not necessarily related.

PATHOLOGIC PICTURE

Strictly clinging to Vinson's definition of the syndrome, there is no pathologic picture. How can there be if there is no organic lesion? Yet those who have found organic lesions of the esophagus describe a pathologic picture. There have been very few autopsies. In a case of my own, to be reported later, the mucous membrane showed thinning of the epithelial layer, with ulceration. The lamina propria and submucosa were replaced by dense collagenous tissue or infiltrated with lymphocytes. There was destruction of the muscularis mucosa. In the muscularis there was moderate fibrosis.

McGee and Goodwin [15] reported an autopsy on a patient who died of hemorrhage. The mucous membrane showed an area where the mucous and submucous layers were broken and denuded. Beneath the mucous membrane was evidence of mild inflammation.

Suzman [16] reported an interesting series of cases. Two patients had had gastroenterostomies. One died from mediastinitis following the passage of a filiform bougie. Autopsy revealed that the mucosa and muscle of the tongue and esophagus showed hyperkeratinization of the epithelium, with areas of desquamation and degenerative atrophic changes in the underlying muscle. The condition did not appear to be inflammatory. Since enough vitamin A was found in the liver, deficiency of vitamin A cannot have been responsible for hyperkeratinization of the epithelium. The presence of areas of mucous membrane simulating leukoplakia and containing immature cells exhibiting

15. McGee, L. C., and Goodwin, T. M.: Ann. Int. Med. **11**:1498, 1938.

16. Suzman, M. M.: Syndrome of Anemia, Glossitis and Dysphagia, Arch. Int. Med. **51**:1 (Jan.) 1933.

mitoses was of interest with respect to the tendency to development of malignant disease. The plexus of Auerbach revealed no abnormality.

From autopsy observations this writer excluded as possibilities for the cause of obstruction: hysteria (Vinson), spasm (Hurst), inflammation of the nerve supply (Kelly) and involvement of the Auerbach plexus (Hurst) similar to achalasia of the cardia; he expressed the view that definite webs, bands or raised folds of mucous membrane are responsible for the obstruction. He also rejected Evans' theory of syphilitic origin. He cited the work of Hill as of interest.

The sequence of anemia and dysphagia is not established. Suzman stated the belief that the manifestations of the syndrome, except for the dysphagia, are indistinguishable from those of idiopathic hypochromic anemia, also called achylic chloranemia, idiopathic secondary anemia, chronic hypochromic anemia and simple achlorhydric anemia, and he stated that the dysphagia is merely a complication of an already well known form of anemia. Hence he recognized no need to regard the Plummer-Vinson syndrome as a separate clinical entity. He stated the belief that the underlying cause is iron deficiency, caused by some gastric abnormality.

A number of authors have stated that there is a close relation to carcinoma of the esophagus, i. e., between the lesions which develop at the upper end of the esophagus in the Plummer-Vinson syndrome and carcinoma in the same location. It is pointed out that both conditions are frequent in women and that the sequence is too frequent to be a coincidence.

According to Ahlbom,[17] 90 per cent of patients with postcricoid carcinoma are women, of which again 90 per cent had had either the Plummer-Vinson syndrome or simple achlorhydric anemia. In the Swedish material, 40 per cent of oral carcinomas are in women; moreover, this author stated that the Plummer-Vinson syndrome is more frequent in Sweden than in other countries. Ahlbom's opinion in this matter is by no means undisputed. It must be remembered that Vinson found no esophageal lesions. He naturally found no connection between these conditions. A number of others also have denied the connection. Among these, Laub [18] has made a special study of the matter in 235 cases of carcinoma of the esophagus. From the histories of these cases he concluded that there is no causal connection between Plummer-Vinson syndrome and carcinoma of the esophagus.

Laub's paper is interesting in a number of other respects. He stated the ages of women with this disease as between 15 and 50 years. The anemia appeared at 18 to 20 years of age; between 20 and 30 the women often lost their teeth, and glossitis later completed the picture.

17. Ahlbom, H. E.: Nord. med. tidskr. **11**:171, 1936.
18. Laub, R.: Acta oto-laryng. **26**:668, 1938.

In one fourth of the cases the spleen was enlarged and the nails were brittle and spoon shaped. Usually only some of these symptoms were noticed in each case.

Laub advanced another hypothesis. In 1 patient of his who died of pneumonia, histologic examination showed atrophy of the thyroid, of the hypophysis and of the pancreas, i. e., pluriglandular insufficiency. This patient, therefore, in addition to the Plummer-Vinson syndrome, as shown by autopsy, had a hormonal disturbance of nearly all the glands of internal secretion. This might mean some relation between the Plummer-Vinson syndrome and glandular disturbances.

A hereditary factor was suggested in a report by McGibbon.[19] He reported the case of a 29 year old woman who had dysphagia and secondary anemia. Esophagoscopy showed a definite postcricoid stricture, which was dilated by bougies. The father of this woman also had a postcricoid web, which was relieved by dilating. His father had died of carcinoma of the esophagus. The interesting features in this report are (1) familial predisposition, the grandfather having died of pharyngeal carcinoma, the son at 62 showing severe dysphagia with anemia and the daughter at 29 revealing the same condition to a lesser degree, and (2) occurrence of dysphagia and anemia in a man, which is very rare. McGibbon observed 3 such cases in four years.

In the case of the daughter there was a decrease of fragility of red cells rather than the increase which other authors have reported. It will be seen from the consideration of the literature on the Plummer-Vinson syndrome that there are many unsettled questions in regard to it. First, is there an organic cause of the dysphagia, or is it of hysterical origin? Second, what is the relation between the dysphagia and the anemia? Third, is there a relation between achlorhydric anemia and the Plummer-Vinson syndrome? Fourth, is there any relation between gastric ulcers and the Plummer-Vinson syndrome? Fifth, is there a relation between the Plummer-Vinson syndrome and carcinoma of the esophagus? Sixth, are all the symptoms, dysphagia, glossitis, anemia and achlorhydria, merely different phases of one disease, for which some toxic or dietetic factor is responsible? Seventh, what is the causative factor?

There is as yet no clear answer to any of these questions. It may well be that there are two conditions, the hysterical dysphagia described first by Blankenstein and then by Plummer and Vinson, not associated with any organic lesions, and an organic disease associated with anemia or dietary deficiency.

I should like to present the subject as it has come to my attention and to report 2 cases. A number of years ago I performed an esophagoscopic study at the Rockefeller Institute, on a woman of about 50 years

19. McGibbon, J. E. G.: J. Laryng. & Otol. **53**:32, 1938.

who was having difficulty in swallowing. The authorities there stated that she was suffering from a vitamin deficiency disease, which also affected other mucous membranes (mouth, nose, vagina and rectum). Their idea was that in certain cases of dietary deficiency any one of the orifices of the body might show an atrophic lesion of its mucous membrane.

The esophagus showed in its upper part a constriction which seemed to be caused by thin scar tissue smoothly distributed over the whole surface of the mucous membrane. It was very fragile, and the tube slid easily downward. When the tube was withdrawn, this scar tissue was cracked, as if it had lost all elasticity, leaving a number of shallow, bleeding fissures. In one or two places thin, filmlike scar had brushed off, leaving a shallow ulcer.

I have observed a number of similar cases. The history is always of difficulty in swallowing; at times so slight as scarcely to be noticeable, at times more severe. The anemia has never been severe; there has been some apathy and weakness. As a rule the passage of the tube and a subsequent high vitamin diet have relieved the symptoms.

It must be emphasized that the esophageal lesion is at times so slight as not to be suspected till the tube is withdrawn, when the cracks and abrasions make it evident.

I shall now cite 2 cases to illustrate the point that scars and webs may form in the esophagus after operations on the stomach. These may be on a basis of dietary deficiency.

REPORT OF CASES

CASE 1.—A woman of 63 was admitted Sept. 22, 1938 to the Presbyterian Hospital, complaining of gastric pain of ten years' duration. Twenty-six years before she had had the same pain, relieved at that time by dieting. It had been attributed to ulcer of the stomach. This pain recurred ten years before admission, being evident only when the stomach was empty. General physical examination revealed no abnormality; the patient was not anemic. She was operated on. A pyloric ulcer was found, and the affected portion of the stomach was removed. The remaining portion of the stomach was united to the jejunum, and the stump of the duodenum was closed. The third day after the operation she showed signs of a duodenal fistula, which did not close till the eighteenth day after operation. She was discharged from the hospital November 5, about six weeks after admission. During the last three weeks in the hospital she complained of choking spells when she ate and of a lump in her throat.

A month after her discharge she was readmitted for roentgen study of her dysphagia. A stricture was demonstrated at about the level of the second costal cartilage. Roentgen examination of the esophagus on December 13 disclosed a constriction about at the level of the second costicartilage. This constriction was about 8 cm. in length and about 2 mm. in diameter. Above it the esophagus was very slightly dilated. The barium passed through it very slowly. The middle third of the esophagus remained narrow over a distance of about 7 cm. In this area crinkles in the wall were present, suggesting contractions of the circular muscle, such as are occasionally seen. However, these marginal indentations remained

remarkably constant on a series of seven exposures in various degrees of rotation. In the lower 2 or 3 inches (5 to 7.6 cm.) of the esophagus the wall expanded as usual, and the mucosal pattern appeared normal.

The roentgenologist commented: "This is a rather unusual group of findings. The narrow constriction at the second costicartilage might be due to a carcinoma. It might be due to a benign stricture, in view of the dilatation above it. The more extensive narrowing farther down suggests the possibility of esophagitis. This casts further doubt on the possible malignant nature of the upper constriction. It would be desirable to study the lesion from the inside. A couple of films of the stump of the stomach were taken, which show a normally functioning partial gastrectomy. The jejunal mucosal pattern appears normal."

Fig. 1 (case 1).—*A*, partial obstruction of the esophagus ten weeks after gastrectomy. The patient had had difficulty in swallowing for about seven weeks. *B*, complete closure due to the stricture.

The patient's third admission was on Jan. 10, 1939. The dysphagia was at this time so severe that she could swallow nothing. The duodenal fistula had reopened. The basal metabolic rate was low, and general edema from a low level of blood protein was present. A later examination of the esophagus (January 10) was made. "Reexamination of the esophagus disclosed a practically complete obstruction at the site of the narrowed constriction described on December 13, at the level of the second costicartilage. The rapid increase in this constriction is strongly suggestive of carcinoma. The wall was not seen below this level because of the high degree of the obstruction."

She was operated on again (January 11), and a gastrostomy was done for feeding purposes. Esophagoscopy at that time revealed this scar tissue in the mucous membrane of the mouth of the esophagus. Here and there were shallow ulcerations where the scar tissue had been brushed off the walls. Twenty cubic

Fig. 2.—Mucous membrane removed from the espohagus post mortem. There were no gross ulcers. Microscopic examination showed thinning or destruction of the epithelium, the lamina propria and the submucosa being replaced by moderately dense collagenous tissue. The muscularis mucosae had disappeared. There was a small amount of fibrous tissue in the muscularis.

millimeters from the teeth there was an almost complete closure of the lumen, one very small fistula being seen through the ulcerated stricture.

The postoperative course was stormy. She had postoperative pneumonia on the second day and right parotitis on the fourth day. She had edema from a low level of serum protein. Then a septic temperature developed, and a subphrenic abscess was drained. She slowly recovered after this, but the duodenal fistula remanied open, and the gastrostomy was unsatisfactory. Two months after the second operation, esophagoscopy was again performed.

A 9 mm. esophagoscope was passed, and about 3 cm. down it encountered a strictured area in about the same region mentioned before. The walls of the esophagus had changed in this respect; they no longer looked ulcerated. There was a web with a little hole through it. A wire was passed through the stricture, and then the stricture was dilated up to the diameter of a no. 22 French catheter. A rubber feeding tube was passed into the stomach.

The fourth day after this procedure she had pneumonia and died. There was an autopsy. The esophagus was sufficiently dilated in its upper portion, but 15 cm. from the stomach its wall was greatly thickened, and the lumen was much decreased. At this place the mucosa of the esophagus was lost for a distance of 15 cm. The constriction of the lumen was maintained from here to the stomach. In some places the mucosa was intact, and where intact, it was smoother and more glistening than normal. The thickening of the wall was caused by gray, glistening firm tissue.

Microscopic examination showed slightly thinned squamous epithelium. The lamina propria and the submucosa were replaced by moderately dense collagenous tissue. The muscularis mucosae could not be identified. There was a small amount of fibrous tissue in the muscularis.

Comment.—This patient gave a long history of gastric disease, which was found on operation to be an ulcer. The pylorus was resected. She had a duodenal fistula. This could certainly cause dietary deficiency. From no other evident cause extensive ulceration developed, which in healing produced a fibrous web. The same process was evidently at work in the lower end of the esophagus when she died. Such a benign ulceration developing in a case of gastric disease or in any other case of dietary deficiency certainly might be expected to form fibrous webs as it healed. Such a sequence would certainly afford an explanation of webs and bands in the mouth of the esophagus.

Case 2.—A man 43 years old was admitted to the Lenox Hill Hospital Feb. 21, 1928, wtih a diagnosis of ulcer of the stomach. He had had symptoms for seven years. He was operated on February 25, and a large gastric ulcer was found. A gastroenterostomy was done. The man did well and was discharged March 16, 1929; on May 19 he was feeling well except for pain two hours before eating. He was readmitted on Jan. 8, 1938. He had been well till two months before, when there developed (1) gnawing pain in the epigastrium, (2) regurgitation of mucus, (3) loss of weight and (4) occasional vomiting.

The roentgen report on the esophagus and stomach on Jan. 9, 1938 revealed the following data:

"A barium sulfate meal was administered, and a high degree of obstruction was noted in the esophagus, about 2 inches (5 cm.) above the diaphragm. At this point a sharp cutoff in the barium column was noted, and an annular filling defect was

apparently present. The barium passed this obstruction very slowly and filled the stomach. The upper portion of the esophagus showed marked dilatation. A gastroenterostomy has been performed in the region of the midline of the greater curvature, and barium passed immediately through the stomach into the small intestine. A small amount of barium passed out through the pylorus and demonstrated marked deformity of the first portion of the duodenum. The rugal folds in the upper portion of the stomach showed marked swelling and some irregularity. No evidence of a filling defect could be demonstrated here, however."

At six hours the stomach was empty. The meal was in the distal part of the colon, with the head of the column at the descending. The findings would indicate an obstructive lesion in the lower third of the esophagus, probably neoplastic. The gastroenterostomy appeared to be functioning normally. There was marked deformity of the duodenal cap.

Esophagoscopy was done on January 17. The report was as follows: "This is a very interesting case. The normal mucous membrane for the whole length of the esophagus appears to have been destroyed and replaced by a thin, fragile scar

Fig. 3 (case 2).—Complete closure of the esophagus a short distance from the diaphragm.

tissue. The walls of the esophagus have adhered in numerous places, so that stretched across the lumen are weblike adhesions, which were broken as the tube passed down. At the point of the obstruction there is dense scar tissue which almost completely closes the lumen of the esophagus. A small lumen can be seen passing through this, although a small sound could not be passed. The walls of the esophagus are very fragile, and any manipulation should be undertaken with the greatest caution. I suggest passing a filiform bougie into the stomach through the esophagoscope, catching this up through the gastrostomy opening and putting a thread through. This would permit retrograde bougienage."

The warning to undertake any esophageal manipulation with caution was well given. This patient had mediastinitis and died.

Comment.—These 2 cases are not reported as cases of the Plummer-Vinson syndrome. They are, however, reported as showing that there is a connection between gastric disease and atrophic conditions of the esophagus. The first case was obviously a case of dietary deficiency. Evidently the duodenal leakage drained away much of the necessary

nourishment. The ulceration and healing of the esophagus gave an appearance in the upper parts similar to those reported in cases of the Plummer-Vinson syndrome. I cannot think that the esophageal procedure had anything to do with the patient's death. The appearances in the second case may explain the formation of bands and adhesions. There must have been extensive ulceration. The ulcerated folds falling together might adhere. When the folds were subsequently pulled apart, webs and bands would be formed, some even bridging the lumen.

There is no doubt that esophagoscopy caused the mediastinitis and death of the second patient. The tissues of such an esophagus as his, long diseased and very fibrotic, are easily torn. Even without an actual rupture, leakage might be sufficient to cause infection of the mediastinum.

GENERAL SUMMARY

The literature is reviewed, and such reports as seem most illuminating are cited. My personal experience with this condition is given. The anemia has not been prominent in my cases. Two cases of ulceration and healing secondary to gastric disease are described. There is reason to believe that there may have been dietary deficiencies in these cases.

The impression gained is that many cases are incorrectly reported as instances of the Plummer-Vinson syndrome. In the Plummer-Vinson syndrome esophageal lesions are not shown as a cause of dysphagia. In many cases of dysphagia associated with anemia glossitis reported as examples of the Plummer-Vinson syndrome the condition must be something else, a distinct entity, to be sure, but not on a hysterical basis.

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THE SACCULE

OBSERVATIONS ON A DIFFERENTIATED REENFORCED AREA OF THE SACCULAR WALL IN MAN

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Ever since Scarpa [1] described the membranous vesicles in the vestibule of the labyrinth their walls have generally been considered as undifferentiated except for the areas of the maculas. Thus it is customary for modern anatomists, in describing the membranous labyrinth, to include the walls of the utricle, the saccule and the membranous semicircular canals in a short general statement. Such a description has been given by the following authors: Kolmer,[2] A. Eckert Möbius,[3] Shambaugh [4] and Maximow and Bloom.[5] A general agreement exists that these membranous walls are made up of two thin layers of cells separated by a basement membrane, an outer connective tissue or mesothelial layer and an inner layer of exceedingly thin (3 to 4 microns) polygonal squamous cells. Occasional branched melanophores are found in the outer layer of connective tissue, especially in lower mammalian forms.

While examining a large number of sections of human temporal bones I have noted a regular area of thickening of the saccular wall in its anterolateral aspect (fig. 1 *A*). This appears to be a constant feature of the free unattached saccular wall, extending over a limited but apparently constant surface of this wall. Only as an exception may one observe a small circumscribed area of thickening by connective tissue in the thin saccular wall beyond the posterior lip. There is

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From the Division of Otolaryngology, Department of Surgery, University of Chicago.

1. Scarpa, A.: Anatomische Untersuchungen des Gehörs und Geruchs, translated from the Latin, 1797, pp. 76-85.

2. Kolmer, W., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1928, vol. 3, pt. 1, p. 286.

3. Eckert Möbius, A., in Denker, A., and Kahler, O.: Handbuch der Hals, Nasen- Ohren-Heilkunde, Berlin, Julius Springer, 1926, vol. 6, pt. 1, p. 305.

4. Shambaugh, G. E., in Cowdry, E. V.: Special Cytology, ed. 2, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1,359.

5. Maximow, A. A., and Bloom, W.: Textbook of Histology, Philadelphia, W. B. Saunders Company, 1930, pp. 773-774.

also a variation in the thickness of the posterior lip itself, but within narrow limits. In many specimens in which some accidental force reacted with the saccule, a differential response from the reenforced area of saccular wall and from the thin area was observed. The area of thickened saccular wall showed greater resistance to pressure from without, and a partial collapse of the remaining saccular wall was striking, as it stopped at the junction between the reenforced and the

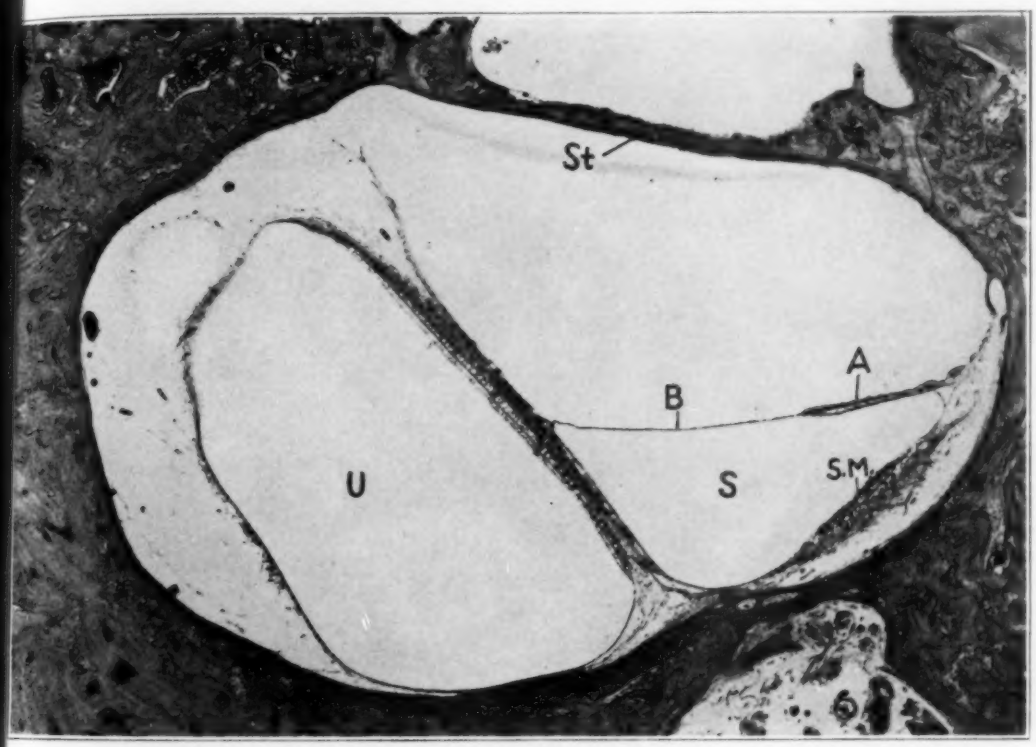

Fig. 1.—Photomicrograph (× about 25) of a horizontal section through the vestibule of the human labyrinth, with no evidence of distortion of the utricle (*U*) or saccule (*S*), and illustrating the relation of the reenforced area of saccular wall (*A*) to the nonreenforced thin surface (*B*) continuous with it and to the saccular macula (*S. M.*) below. *St* is the stapes footplate.

thin surface (fig. 2). When rupture of the saccular wall occurred, for some reason or other it was practically always at the junction of the reenforced area and the thin area (figs. 3 and 4).

Examination of other mammalian saccules for this differentiated area of thickening was performed, but results of that made on the cat, the

dog (fig. 5), the guinea pig and the rabbit were negative. In the monkey, Macacus rhesus, a definite differentiation of the free saccular wall was found (fig. 6), but this appears to have a different shape, extending in equal width from the anterolateral and from the posterolateral (utricular) aspect of the superior pole of the saccule. Furthermore, this area of thickening of the saccular wall in man can be seen in pyroxylin (celloidin) casts of the labyrinth, both in the unstained

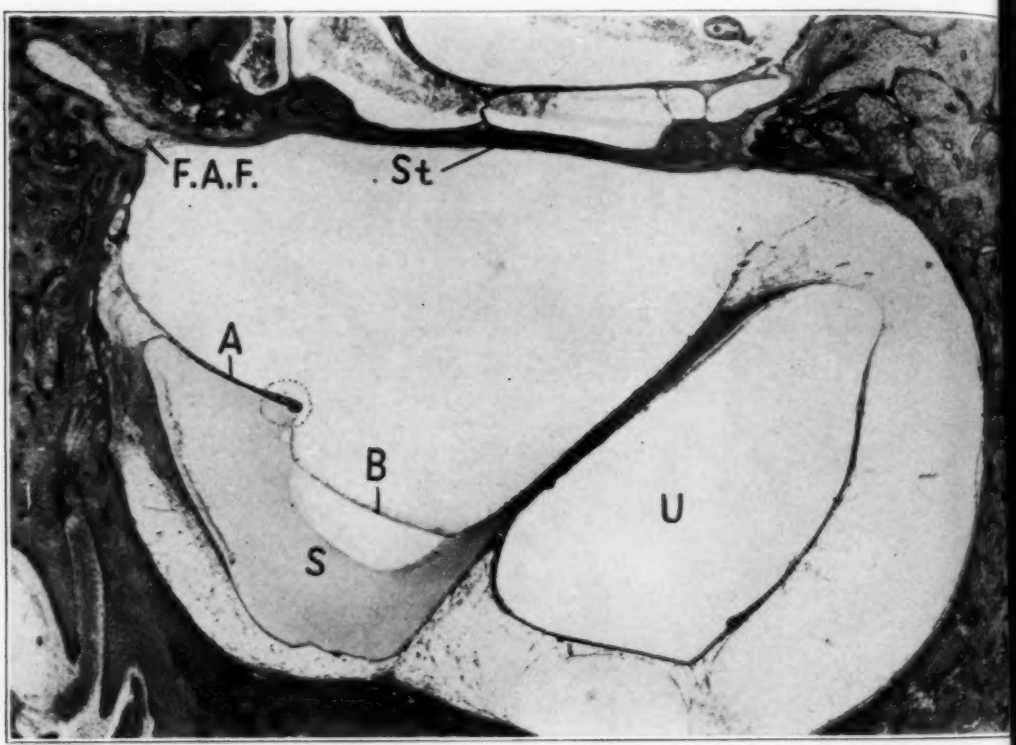

Fig. 2.—Photomicrograph (× about 25) of a horizontal section through the vestibule, showing the differential response of the saccular wall in its anterior thickened area (*A*) and the adjacent thin area (*B*). The collapse of the thin wall of the saccule is seen to begin abruptly where it joins the reenforced area. The lip of the reenforced part appears to resist this collapsing force. *S* represents the saccule, *U* the utricle, *St* the stapes footplate and *F. A. F.* the fissula ante fenestrum. The area within the circle is illustrated in figure 11, a drawing.

specimens and in those stained with osmic acid. In one of the latter preparations blood vessels could be seen within the area, one vessel running lengthwise along the posterior lip of this area where it meets the thin part of the saccular wall.

The membranous labyrinth in the vestibule can be exposed by a razor blade section of a decalcified fetal bone made in the plane of the stapes footplate but just superior to it. In such a preparation (fig. 7) the utricle is seen well distended, the saccule partially collapsed through its thin area, the area of thickening on the anterolateral wall of the saccule being thus brought into relief. As such a preparation dries, the saccular wall is seen to tear along the line of junction between the

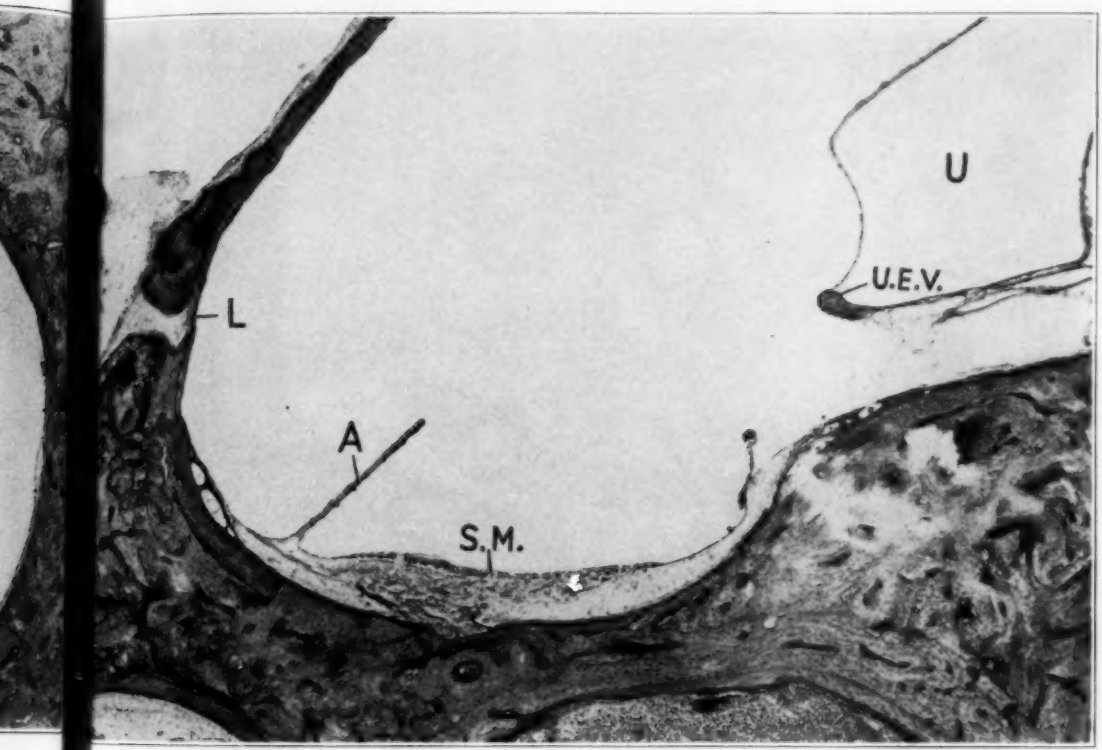

Fig. 3.—Photomicrograph (× about 25) of a horizontal section through the vestibule of the human labyrinth, showing rupture of the thin saccular wall while the reenforced surface (*A*) of the saccule remains intact. *U* is the utricle and *U.E.V.* the utriculoendolymphatic fold or valve. *L* is the anterior part of the annular ligament and *S.M.* the saccular macula.

thin and the reenforced area of saccular wall, and the saccule collapses except for this anterolateral surface. This surface is made crescent shaped by connective tissue fibers from the upper part of the saccule coursing out to the macular area of the utricle, and the noncollapsed wall extends down to the wall of the vestibule near the ductus reuniens.

A wax plate model [6] of the human saccule was made in order to show the extent of this differentiated area of the saccular wall. The area of the saccular macula was also indicated. The reconstruction of the saccule was made at thirty-seven diameters from sections of a 27 year old man. Examination of the model of the saccule (fig. 8) shows the reenforced area to be largely a triangular surface with the apex down, occupying the anterior part of the lateral wall of the saccule. The apex

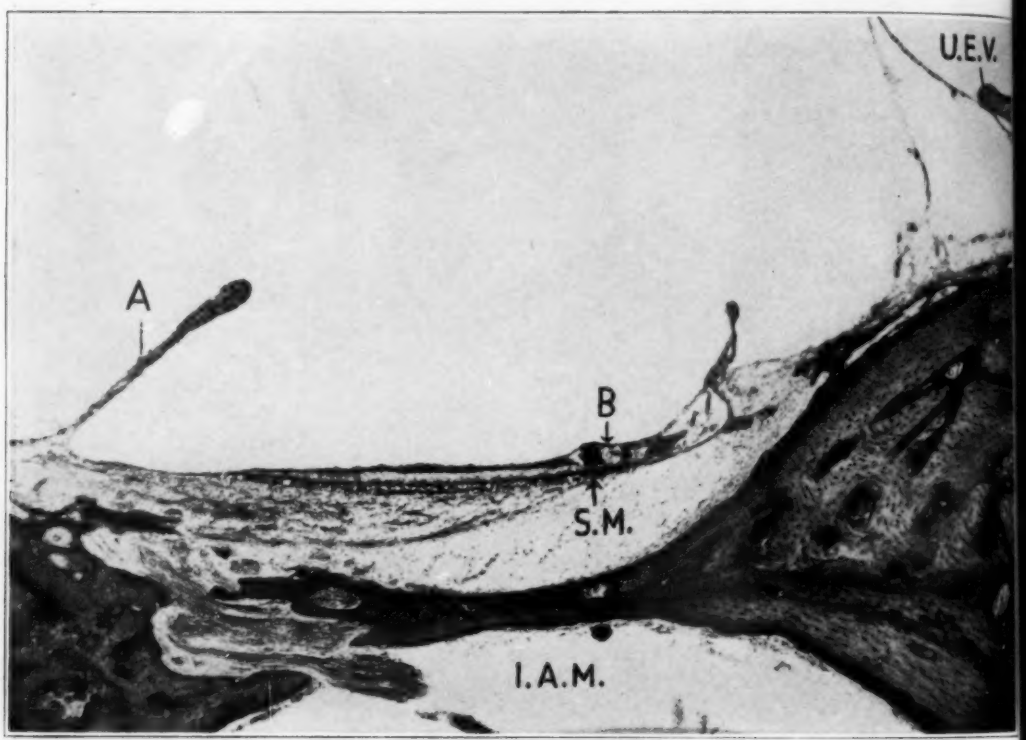

Fig. 4.—Photomicrograph (× about 50) of a horizontal section through a ruptured saccule, illustrating the resistance of the reenforced surface (*A*) of the saccule to collapse or tearing. A portion of the collapsed thin saccular wall (*B*) lies on the saccular macula (*S. M.*). At *U. E. V.* is the utriculoendolymphatic fold or valve and at *I. A. M.* the internal acoustic meatus.

of this differentiated area begins at the level of the saccular duct and at that of the lower limit of the saccular macula. The widest part is at

6. Dr. G. Bartelmez helped me with the construction of the model, and Miss Agnes Nixon made the drawings.

the level where the saccular macula begins to bend posterolaterally from a vertical plane. Above this level the reenforced area narrows a little. Histologic examination shows that the lower two thirds of the reenforced saccular wall is thicker than the upper third, particularly along the free, or posterior, edge. The line of junction between this free edge and the continuing thin area of saccular wall is therefore distinct through-

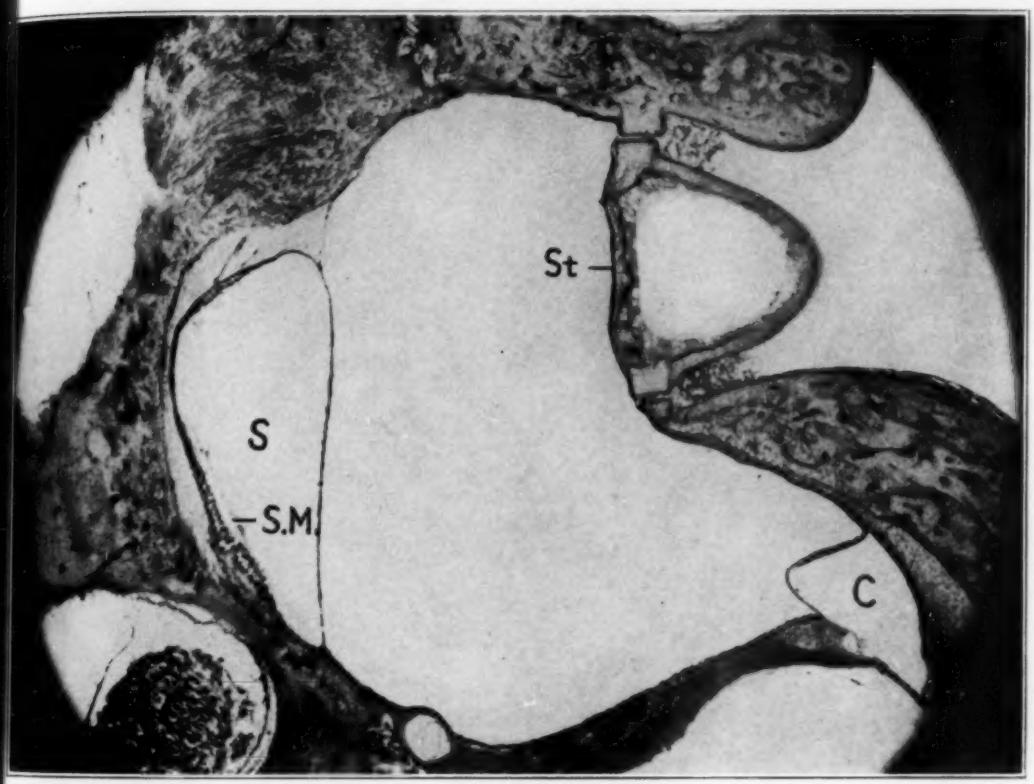

Fig. 5.—Photomicrograph (× about 40) through the vestibule of the labyrinth of a dog, illustrating the absence of a differentiated, or thickened, area in the lateral wall of the saccule (*S*). *S. M.* is the saccular macula, *C* the cochlear duct and *St* the stapes.

out the lower two thirds of the structure. Most of the sections illustrated in this paper are through the middle of the saccule and in a horizontal plane.

The orientation, form and contour of the saccular macula can be seen in figure 9. At the superior pole of the saccule in the region where

it is in relation to the utricle, many connective tissue bundles course in the perilymph to connect the walls of the utricle and saccule and the walls of the saccule to the upper area of the vestibular wall in the spherical recess, but these bundles are easily differentiated in the histologic sections from the thickened area of the saccular wall itself. Some of these connective tissue bundles extend from the upper area of thickening of the saccular wall to the macular area of the utricle and course

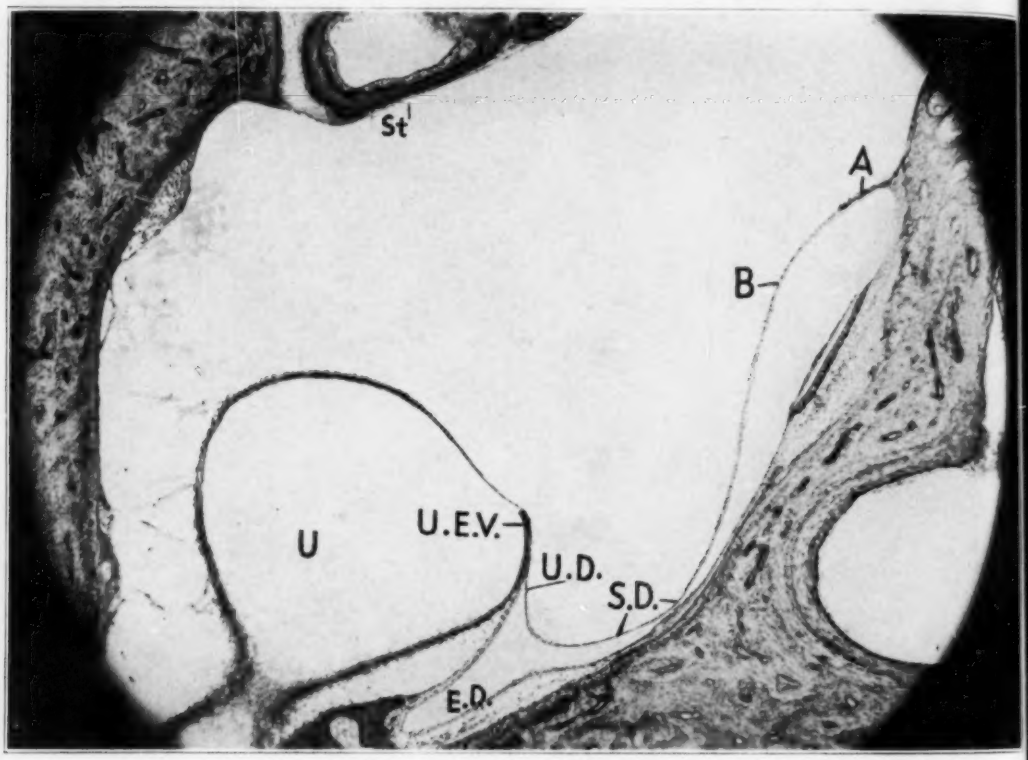

Fig. 6.—Photomicrograph (× about 42) of a section through the lower level of the vestibule of the labyrinth of a monkey (Macacus rhesus), showing the thickened portion of saccular wall (*A*) comparable to that seen in the sections of the human labyrinth at a corresponding level. The thin portion of the saccular wall (*B*) continues into the formation of the saccular duct (*S. D.*). *U* is the utricle, and the utriculoendolymphatic fold or valve is clearly seen at *U. E. V.* The utricular duct is indicated (*U. D.*) and the continuation of the saccular and the utricular duct as the endolymphatic duct (*E. D.*) *St* is the stapes footplate.

free in the perilymphatic space. These help to give a crescent-shaped appearance to the noncollapsed saccular wall seen in the photograph

of the especially prepared wet specimen showing the collapse of the membranous saccule (fig. 7).

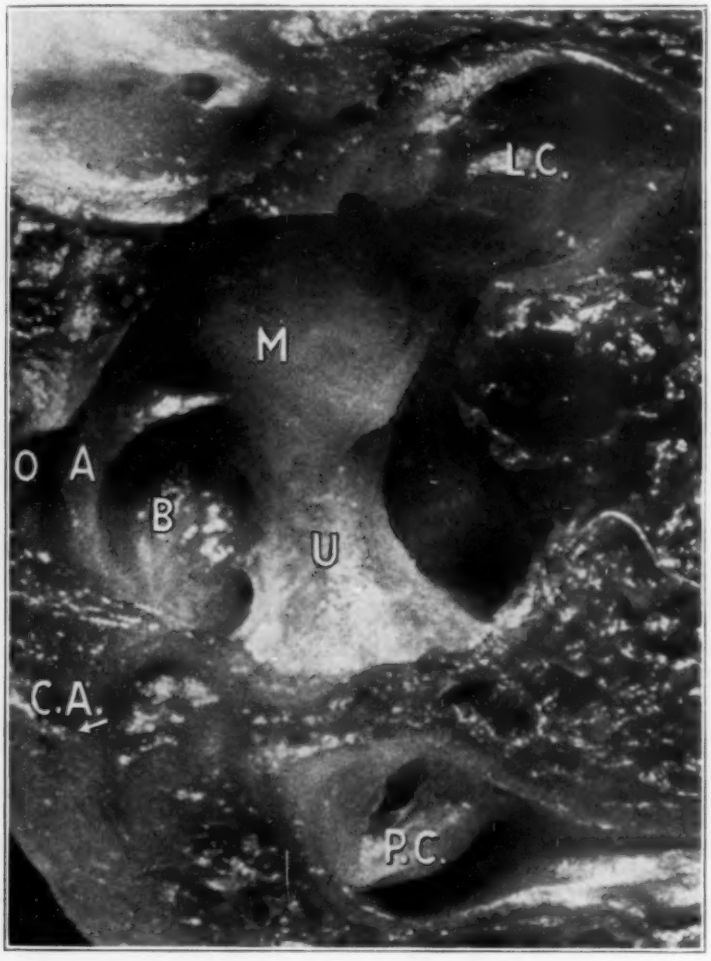

Fig. 7.—Photograph (enlarged, × 14) of the decalcified fetal temporal bone in its inferolateral aspect, prepared to show the membranous labyrinth within and adjacent to the vestibule. The thin wall of the saccule (*B*) has collapsed and throws into relief the reenforced area of the saccular wall (*A*). The utricle (*U*) is distended, and the nerve fibers to its macula are seen at *M*; *L.C.* and *P.C.* are the membranous ampullae of the horizontal canal and the posterior vertical canal respectively. At *C.A.* is the mouth of the cochlear aqueduct and at *O* the anterior edge of the oval window.

Note: There is difference in the orientation of the saccula in the fetus and in the adult with respect to the plane of the oval window: In the fetal bone the oval window is almost in a horizontal plane while in the adult it is almost in a vertical plane.

A large part of the posterosuperior wall of the saccule is attached to the utricle lying above it. This attachment is made by connective tissue elements lying between the walls of the saccule and utricle where they are opposed. These connective tissue elements extend to the wall of the vestibule. This firm attachment of a portion of the saccular wall to the heavier utricular wall in the area beneath the utricular macula acts as a support that resists shrinking forces affecting the volume of the saccule, as in fixation. A small degree of this

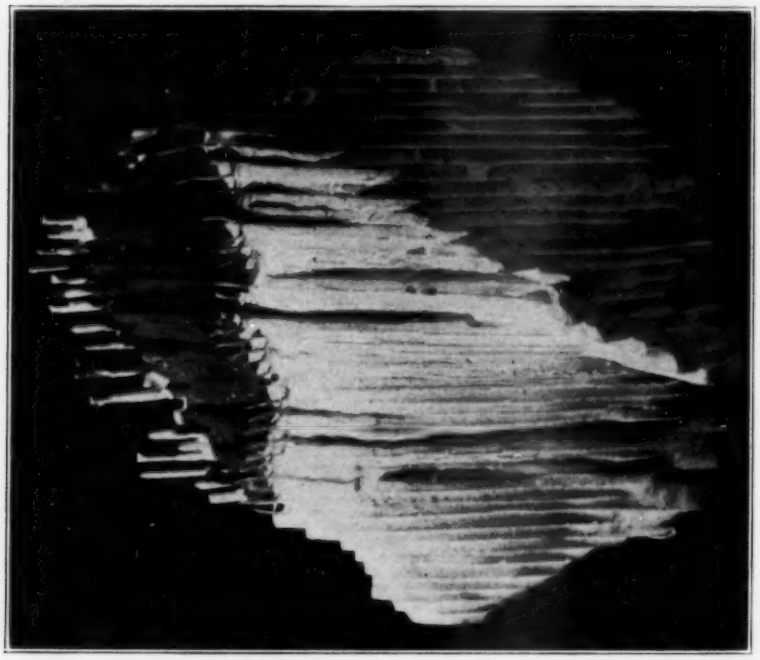

Fig. 8.—Photograph of the reconstruction model (full size) of the saccule in its lateral aspect, with the area of reenforced saccular wall in black, outlined by the metal rods. The drawings (figs. 9, 10 and 11) explain the orientation of the rest of the model.

shrinkage of the saccule has taken place about the inferoposterior aspect of this attached area in the specimen from which the model was made, and appears in the photograph (fig. 8) and the drawing (fig. 10) of the model.

In human material it is extremely difficult to preserve completely distended and intact all of the membranous labyrinth, particularly those structures lying within the vestibule. Hematoxylin and eosin stains

of the saccular wall in the reenforced area indicate that the reenforcement, or thickening, is due to an increase of the connective tissue, an extension into the saccular wall of the connective tissue holding the saccular wall in this region to the anterior bony wall of the vestibule, and with such stains small blood vessels can be seen particularly near the line of junction between the thickened area and the thin area of

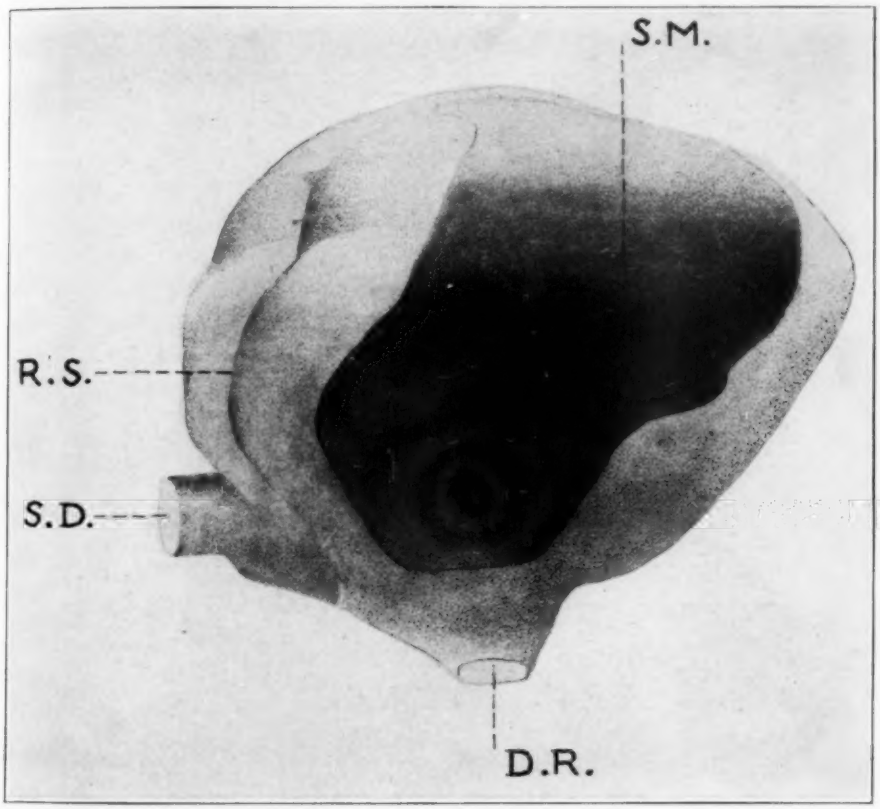

Fig. 9.—A full scale drawing from the reconstructed model of the saccule in its anterior aspect (as viewed from within the internal acoustic meatus). The entire saccular macula (*S. M.*) is outlined and shaded to show how its superior portion is bent posteriorly. *R. S.* represents the portion of the saccule attached to the medial lip of the spherical recess of the bony vestibule. *S. D.* indicates the beginning of the saccular duct and *D. R.* the beginning of the ductus reuniens.

the wall of the saccule. The epithelial and the mesothelial layer of the saccule do not reduce silver in Bielschowsky preparations; thus in the thin area of the saccular wall they appear as one clear layer, while in the thick area these layers separate to cover the saccular and the

vestibular surface of the thickened wall and have between them a mass of connective tissue elements and collagen fibrils that reduce silver (fig. 11). An azocarmine stain of this area adds no new information.

This thickening of the saccular wall undoubtedly has been observed by many working with temporal bone sections, but I have never seen it mentioned. However, a number of photomicrographs in existing articles show this differentiation of the saccular wall clearly.[7]

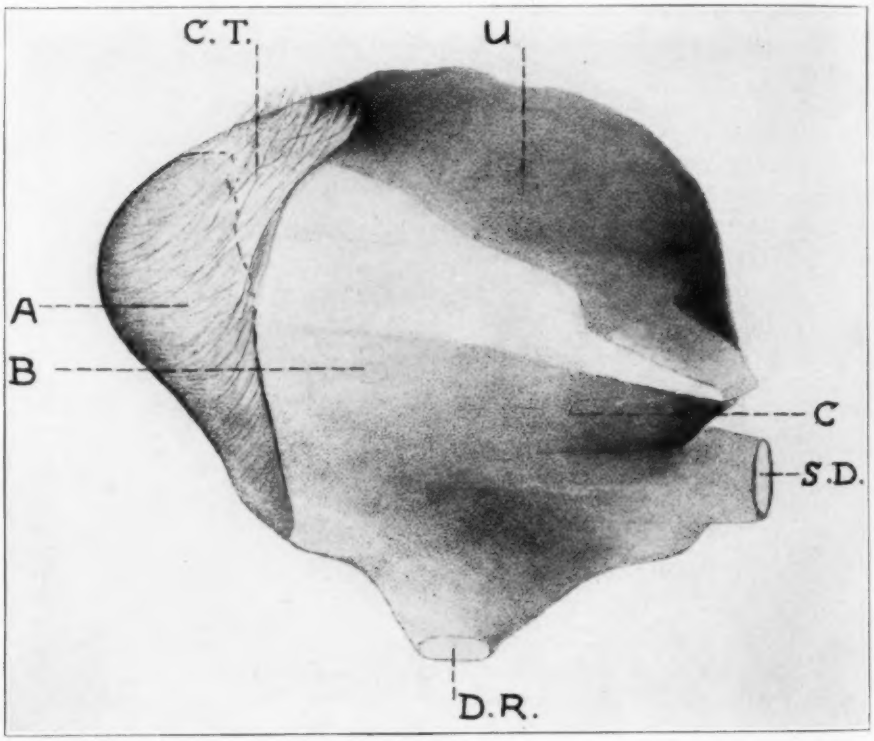

Fig. 10.—A full scale drawing from the reconstructed model of the saccule in its superolateral aspect. The area of reenforced saccular wall is indicated by the shaded area in the anterior aspect (*A*). The thin part of the saccular wall is indicated by *B*. *C.T.* represents connective tissue elements coursing from the upper part of the reenforced saccular wall to the utricle, *U* the upper posterior surface of the saccule in contact with the utricle and *C* the partially collapsed portion of the saccule immediately below its attachment to the utricle. *S.D.* is the beginning of the saccular duct and *D.R.* the beginning of the ductus reuniens.

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7. (*a*) Kolmer,[2] p. 287, fig. 35. (*b*) Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1923, vol. 1, p. 789, fig. 282. (*c*) Guild, S. R.: Early Stages of Otosclerosis [figs. 18 and 21], Arch. Otolaryng. **12**:457-483 (Oct.) 1930.

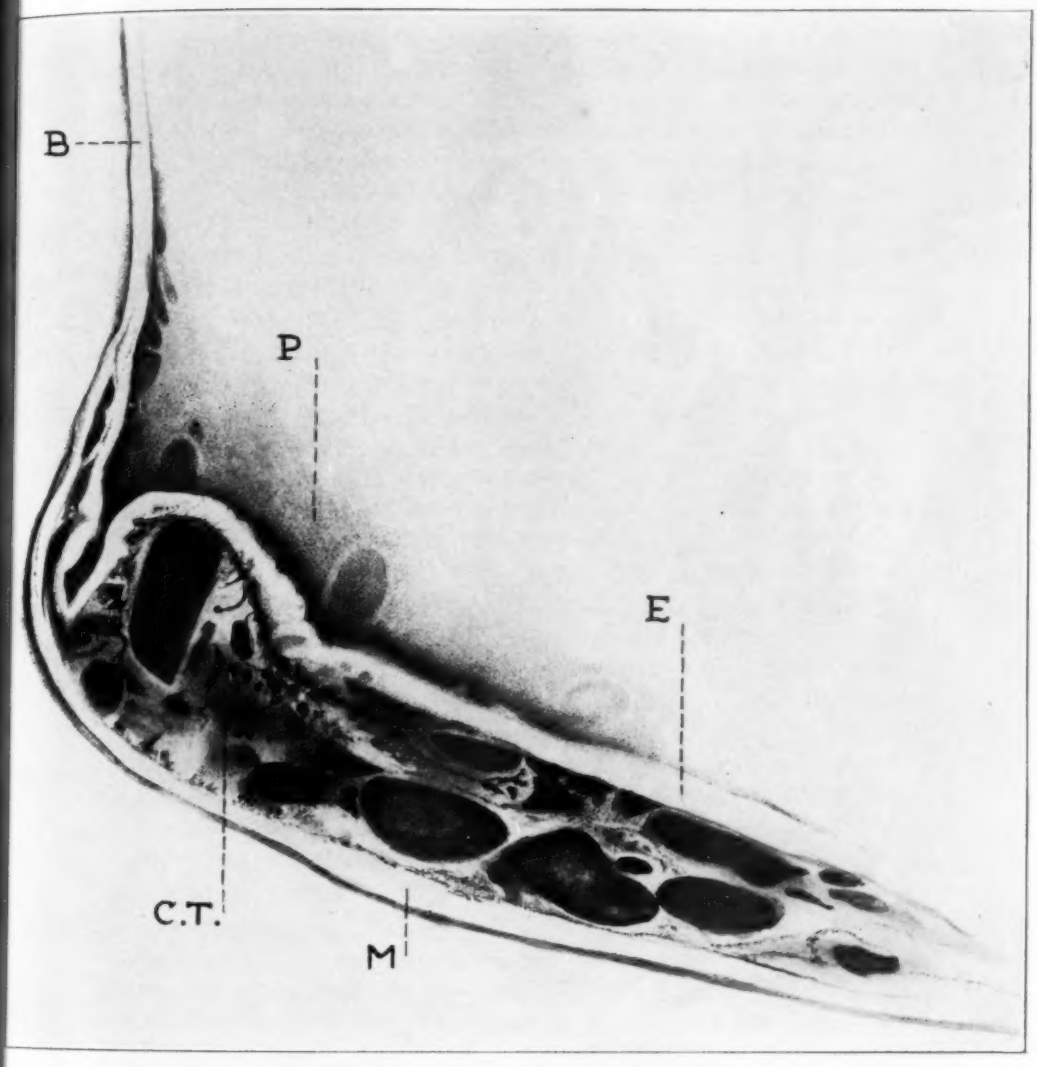

Fig. 11.—Drawing from a section (× 375) stained by Bielschowsky's method. This section is through the saccular wall at the junction between the reenforced and the thin portion (the area inclosed by a circle in fig. 2). The thin part of the saccular wall, composed of a mesothelial and an epithelial layer, does not reduce silver and thus appears as one clear layer. The reenforced area of the saccular wall is seen to be limited by two clear layers; one is the mesothelial layer (*M*) on the outer surface of the saccule and the other the epithelial layer (*E*) on the inner surface of the saccule. Between them are connective tissue and collagen fibrils which reduce silver and are thus in marked contrast to the surface layers. *P* is precipitated silver lying against the saccular wall.

COMMENT

Some functional significance may be attributed to this anatomic structure.

The presence of a small but constant area of differentiated, thickened saccular wall in man and its absence in the lower mammalian forms examined suggest that the saccule in man may have a function different from that in lower forms. In general the function of the saccule is not clearly understood. Investigations carried out on lower mammalian forms, on amphibians and on fish, strongly suggest that it has an acoustic function. In these forms, in which the wall of the saccule is uniformly thin, the macula may respond to acoustic as well as static pressure changes.

Examination of the reconstructed model of the saccule reveals that the medial lip of the recess sphericus and the reenforced anterolateral membranous wall of the saccule resemble the sides of a deep saucer, at the bottom of which rests the saccular macula. This orientation of the saccular macula may have a protective purpose, shielding the macula from disturbances originating at the oval window. It is of interest in this regard to consider the investigations on the motion of the stapes in man (von Békésy;[8] Stuhlman Jr.[9]). These indicate that a rocking motion (trap door action) around a vertical axis through the posterior edge of the footplate is the normal physiologic one, the anterior edge of the stapes undergoing the greatest displacement while the posterior edge is practically fixed. Now examination of the model of the saccule with the stapes represented in its anatomic relation to it reveals that disturbances arising from these movements of the anterior part of the stapes footplate would impinge on the reenforced saccular wall before reaching the saccular macula. This suggests that the saccular macula in man may be protected from acoustic oscillations by this reenforced portion of the saccular wall in contrast to the saccular macula in lower forms. Thus the experimental findings in these lower forms of an acoustic function for the saccule may not hold for man or the monkey (Macacus rhesus). When the displacement of the conducting mechanism is increased, as by acoustic stimuli above the threshold of pain, there is evidence that the stapes footplate moves in a different way (rotating about a horizontal axis through the length of the stapes).[8] Under these conditions, as well as with slow but large static pressure changes, a differential reaction of the saccular macula may be produced by the differential response of the reenforced and the thin area of saccular wall. In any event, it would be more reasonable to expect from

8. von Békésy, G.: Akust. Ztschr. **1**:13-24, 1936.

9. Stuhlman, O., Jr.: J. Acoust. Soc. America **9**:119-128 (Oct.) 1937.

my observations that this differentiated area of saccular wall in man has a functional significance and that it does not represent merely a support for a few extremely small blood vessels.

SUMMARY

A clearly defined, small, triangular-crescent-shaped area of reenforcement of the saccular wall is described. It is found to be a constant anatomic feature of the human saccule in its anterolateral aspect. This reenforcement is due to connective tissue entering from the margins of the vestibule and separating the epithelial and the mesothelial layer of the saccular wall. A structure resembling it is found in the monkey but in no lower mammalian forms examined. An enlarged photograph of an especially prepared wet specimen of a temporal bone from a human fetus shows the differential response of the thin and the thick area of saccular wall to forces causing collapse of the saccule. A photograph and drawings of a reconstructed model show the saccular macula and the extent of the differentiated area of the saccular wall. A number of photomicrographs of sections through the saccule are presented to show the general microscopic appearance of the normal saccular wall in man, in the monkey and in the dog and the differential reaction by the reenforced area of the saccular wall in man to forces producing pressure against and rupture of the saccule. The possibility of a functional importance of this anatomic structure is considered.

CANCER OF THE NASOPHARYNX

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Cancer of the nasopharynx, although not the most frequent, is one of the most malignant growths of the upper respiratory and alimentary tracts. Since the medical literature prior to 1900 contains so few references to this tumor, one is led to conclude that it was not clearly recognized as an anatomic entity before about the beginning of the present century.[1] Even today it is probable that in a high percentage of cases it is not correctly diagnosed because of the absence of early symptoms referable to the nasopharynx. Moreover, as will be discussed in greater detail, the later symptoms of the metastases may so overshadow those of the primary tumor that many patients probably die of widely disseminated cancer with the actual site of the primary lesion in the nasopharynx unsuspected. In a cancer clinic about one half of the patients with this disease are referred because of symptoms or objective findings of the metastases alone without a correct diagnosis as to the location of the primary lesion.

The present report is based on observations at the Memorial Hospital for the Treatment of Cancer and Allied Diseases during the past fifteen years and a statistical analysis of the cases of 87 consecutive patients with nasopharyngeal cancer admitted during the years 1930-1934, inclusive.

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1. C. Jackson (Primary Carcinoma of the Nasopharynx: A Table of Cases, J. A. M. A. **37**:371 [Aug. 10] 1901), in 1901, reviewed the indexes of English, French, German and Italian medical literature and stated that he found only 13 cases of cancer of the nasopharynx, to which he added 1 of his own. The earliest case report which he found was that made by Durand-Fardel in 1837. It is of interest that G. E. Smith and W. R. Dawson (Egyptian Mummies, London, George Allen & Unwin, Ltd., 1924) reported finding erosion of the base of the skull in a Byzantine mummy and stated the belief that it was probably due to nasopharyngeal cancer.

PRELIMINARY CONSIDERATIONS

Definition.—The term cancer of the nasopharynx includes all malignant growths arising on the walls of this cavity. Cancer in this area occurs most often on the posterior wall, in the region of the nasopharyngeal tonsil, with its lateral extensions into the recessus pharyngis (fossae of Rosenmüller), and next in frequency on the lateral walls, on the ridge (torus tubarius) which surrounds the orifice of the eustachian tube. Occasionally a growth may originate somewhat lower on the posterior wall, near the junction of the nasal and oral pharynges. The floor of the nasopharynx (the upper surface of the soft palate) and the anterior wall (the posterior edge of the nasal septum) practically never give rise to cancer.

Anatomy of the Nasopharynx.—Morphologically and developmentally the nasopharynx is part of the true pharynx, but functionally it is an annex to the respiratory portion of the nasal cavities. Both anatomically and functionally it is distinctly marked off from the digestive tract. The cavity is irregular and is enclosed by the anterior and posterior and the two lateral walls, the floor and the roof.

The posterior wall, which is nearly vertical, is formed by the mucous membrane covering the aponeurosis of the superior constrictor muscle overlying the anterior portion of the basilar process of the occipital bone and its junction with the body of the sphenoid. The posterior wall contains the pharyngeal tonsil (adenoid), an irregular mass of lymphoid tissue which extends laterally into the recessus pharyngis on each side at the junction of the posterior and lateral walls. In the midline of the pharyngeal tonsil there is found consistently in the child and occasionally in the adult a small recess which runs upward and backward in the wall of the pharynx, known as the pharyngeal bursa. This structure is sometimes in the form of a definite groove, which gives prominence to the lymphoid tissue on each side and may make it appear to be a new growth of tissue.

The anterior wall is formed by the posterior edge of the nasal septum and the posterior nares, just anterior to which are the posterior ends of the middle and inferior turbinates. Growths arising in the posterior portions of the nasal cavity (chiefly the turbinates) often extend into the nasopharynx and may erroneously be included under the designation of nasopharyngeal growths.

The lateral walls are irregular and contain on each side the opening of the eustachian tube, which is surrounded by a posteriorly prominent ridge, the torus tubarius. Between this posterior prominence and the posterior nasopharyngeal wall on each side is the lateral recess, or fossa of Rosenmüller. The fossae of Rosenmüller are of particular significance in nasopharyngeal cancer, since they either give rise to the primary lesion or are eventually invaded in practically all cases. The upper, or anterior, portion of the groove lies directly over the foramen lacerum, which gives direct access to the intracranial cavity and, as will be described presently, favors the early and frequent local intracranial extension of the growth.

The roof of the nasopharynx lies under the body of the sphenoid and on each side extends downward to the superior margin of the orifice of the eustachian tube. The floor of the nasal cavity is formed by the upper surface of the soft palate and the opening into the oral pharynx.

The pharyngeal tonsil or adenoid, an irregular accumulation of lymphoid tissue found consistently in the child, is readily distinguishable, though atrophied, in most adults. This lymphoid tissue usually extends into the fossae of Rosenmüller and occasionally onto the lateral walls into the region of the eustachian opening. Cancer arises most often in areas where this lymphoid tissue is present, and moderate persistence or benign hypertrophy of the pharyngeal tonsil in the adult may closely simulate cancer in appearance.

Lymphatics of the Nasopharynx.—The lymphatics of the nasopharynx are numerous and originate mainly in the pharyngeal tonsil (adenoid). The collecting trunks combine and run laterally and downward on the pharyngeal aponeurosis, some of them terminating in the median and lateral retropharyngeal lymph nodes (fig. 1). Other collecting trunks pass by the retropharyngeal nodes and are then

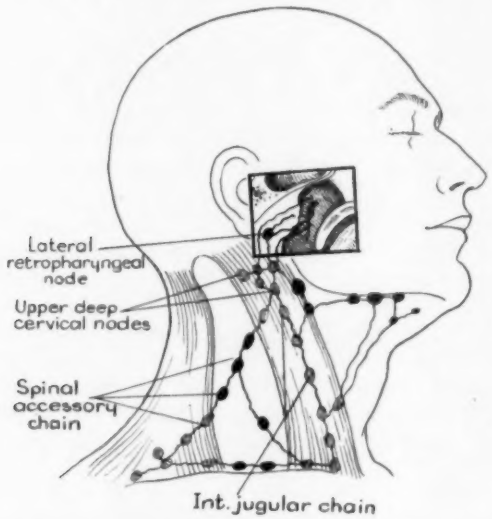

Fig. 1.—Diagram of lymphatics of the nasopharynx (modified after Rouvière). The collecting lymph trunks in the nasopharynx pass in part through the retropharyngeal node but mainly directly to the upper deep cervical lymphatics and then drain downward along either the internal jugular or the spinal accessory chain.

joined by the lymph vessels emerging from these nodes. Both sets then terminate for the most part in the upper deep cervical nodes which are grouped about the upper portion of the internal jugular vein. From these upper deep cervical nodes some efferent branches run down to the middle and lower groups of the anterior chain which lies along the internal jugular vein. Other branches leave the upper deep cervical nodes and run downward along the posterior or spinal accessory chain of cervical lymph nodes following the general course of the eleventh cranial nerve. Although not emphasized by anatomists, it is obvious that the cervical lymphatic system must contain many by-passes which provide routes through which metastases may leave the primary lesion in the nasopharynx or elsewhere and appear first in a lymph node in the middle or lower portion of the neck.

INCIDENCE AND ETIOLOGY

General Incidence.—Cancer of the nasopharynx is undoubtedly much more common than reports in the literature indicate. According to Lack,[2] Herzfeld found only 1 nasopharyngeal tumor in 28,000 cases of cancer; Finder calculated 5 in 40,000 and Gurlt 18 in 10,000. Jackson reported Schmidt as having found no case of nasopharyngeal cancer in 32,997 examinations of the nose and throat. In the light of present day knowledge, these reports, of over twenty-five years ago, indicate that this form of the disease was not well recognized then.

It is unfortunate that in so many reports today nasopharyngeal cancer is included with growths of the nasal cavity and of the pharynx as a whole. This lumping together of tumors from adjacent areas renders the figures in even the best reports unsuitable for statistical analysis with regard to the nasopharynx itself. In the Head and Neck Clinic at the Memorial Hospital cancer of the nasopharynx makes up about 2 per cent of all malignant growths, as compared with such other fairly common varieties as cancer of the palatine tonsil (3 per cent) and of the tongue (7 per cent). In the mortality records of the city of New York from 1936 to 1938, inclusive, cancer of the nasopharynx accounted for only 0.0017 per cent of all deaths from cancer,[3] which is evidence for the assertion that many patients probably die of this disease with the primary lesion undiagnosed.

Age and Sex.—Cancer of the nasopharynx occurs at an earlier age and is found more often in children and at ages below 30 than any other malignant growth of the upper respiratory and alimentary tracts. In a five year period at the Memorial Hospital, 8 patients (9 per cent) were under the age of 15, and 18 per cent of the whole group were under the age of 30. In the series of 87 cases analyzed for this report, the mean age was 45 years, as compared with about 56 to 58 for cancer of such adjacent regions as the tongue, lip, cheek, tonsil and larynx. The highest incidence in any decade fell between 40 and 49 (24 per cent). The youngest patient was 4 years of age; the oldest, 72. Trotter[4] reported that the majority of his patients were between the ages of 18 and 35, but we have found no other such records in the literature.

As with most other forms of cancer of the upper respiratory and alimentary tracts, nasopharyngeal cancer is predominantly a disease of the male (about 80 per cent in our series). The averages in most

2. Lack, H. L.: The Diseases of the Nose and Throat and Its Accessory Sinuses, London, Longmans, Green & Co., 1906.

3. These figures were calculated at our request by Mr. Thomas J. Duffield, Registrar of Records, New York city.

4. Trotter, W.: On Certain Clinically Obscure Malignant Tumors of the Nasopharyngeal Wall, Brit. M. J. **2**:1057, 1911.

reports are similar to this,[5] although Gerlings and den Hoed [6] reported that only 64 per cent of the tumors in their series occurred in males.

Racial Incidence.—Some of the largest series of nasopharyngeal cancer have been reported from China,[7] and many of the investigators have recognized an unusual racial susceptibility of the Chinese to this disease. Digby, Thomas and Tse [8] in reporting 103 cases stated that it is not unusual to have 4 or 5 patients at the same time in a service of forty-two surgical beds in a Hong Kong hospital. Dunlap [7b] from Shanghai reported having seen 16 such lesions in Chinese patients during six years of private practice, although none occurred among the Occidentals who made up 60 per cent of his patients. He also cited a communication from Changsha giving the incidence of nasopharyngeal tumors as about 5 per cent of all cancer. Bonne [9] has noted the peculiar frequency of apparently primary malignant growths of the cervical lymph nodes in Java, Singapore, Sumatra, the Philippine Islands, Indo-China and other parts of the Far East. Although he reported that sometimes there are also lesions in the nasopharynx which are discovered late in the course of the disease, he stated, nevertheless, that the presence of the latter does not necessarily prove their primary nature. He mentioned 3 Chinese patients, 1 of whom had "secondary nasopharyngeal involvement." In our opinion, the whole question might reasonably be explained on the basis of undiscovered primary nasopharyngeal cancer in most of the cases.

Dobson,[10] in commenting on this racial susceptibility, suggested as a caustive factor the accumulated smoke from kerosene lamps, fireplaces, tobacco and candles in the poorly ventilated native Chinese houses. However, Chu,[11] in a general practice limited largely to the Chinese settlement in the city of New York, has seen 11 Chinese with nasopharyngeal cancer, most of whom were born in China but had lived the greater portion of their lives in the United States. The fact that the disease occurs with such frequency in Orientals who live in a fairly

5. (*a*) Needles, W.: Malignant Tumors of the Nasopharynx, J. Nerv. & Ment. Dis. **86**:373, 1937. (*b*) Schinz, H. R.; Zuppinger, A., and Nager, F.: Zürcher Erfahrungen der Radiotherapie von Hypopharynx- und Larynxkrebsen; nebst einem Beitrag zur chirurgischen Therapie des inneren Larynxcarcinoms, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:134, 1936.

6. Gerlings, P. G., and den Hoed, D.: Diagnosis and Therapy of Malignant Tumors of the Nasopharynx, Nederl. tijdschr. v. geneesk. **81**:587, 1937.

7. (*a*) Digby, K. H., and Khoo, F. Y.: Nasopharyngeal Carcinoma, Far East. A. Trop. Med., Tr. Ninth Cong. **2**:903, 1934. (*b*) Dunlap, A. M.: Malignancy of the Nasopharynx and Eustachian Tube, Chinese M J. **53**:68, 1938.

8. Digby, K. H.; Thomas, G. H., and Tse, H. S.: Notes on Carcinoma of the Nasopharynx, Caduceus, 1930, vol. 9, no. 2.

9. Bonne, C.: Cancer and Human Races, Am. J. Cancer **30**:435, 1937.

10. Dobson, W. H.: Cervical Lympho-Sarcoma, China M. J. **38**:786, 1924.

11. Chu, F.: Personal communication to the authors.

normal American environment suggests that the tendency is racially inherited and not due to either hygienic or dietary factors.

Causative Factors.—An analysis of the present series reveals no consistent or conclusive evidence of a specific causative factor. The nasopharynx is not subject to the more common forms of chronic irritation which are known to produce definite precancerous changes in the oral mucous membranes. So far as we know, leukoplakia is never found in the nasopharynx. Tobacco could obviously cause no local changes unless the smoke were inhaled and exhaled through the nose or unless snuff were used. The incidence of smokers in our series was no greater than the normal percentage of addiction in healthy men, and none of our patients took snuff. Routine Wassermann tests were performed in 37 of our cases, of which 4 (about 10 per cent) were positive. Although this figure is slightly higher than the average incidence, the number of cases is too small to permit of definite conclusions regarding the etiologic significance of syphilis.

Other forms of chronic irritation which might possibly affect the nasopharynx are chronic infection or unusual susceptibility to colds or allergy. So far as we could determine, however, the incidence of frequent infections of the upper respiratory tract in our patients was low (less than 10 per cent). Since nasopharyngeal cancer arises so often in the pharyngeal tonsil, one might look for an associated hypertrophy or other abnormality of all the lymphoid tissues in Waldemeyer's ring, but our cases failed to reveal an unusually high incidence in this regard.

For these reasons we are in accord with most investigators that it seems impossible to demonstrate any specific etiologic factors for nasopharyngeal cancer.

PATHOLOGY

A histologic classification of the growths in the series herein reported is given in table 1. Since cancer of the nasopharynx arises most commonly at the site of the pharyngeal tonsil, it is natural that highly anaplastic epidermoid carcinomas and lymphosarcomas should make up a large proportion of the total, as is also found with the palatine tonsil. Minor salivary glands are present in the mucous membrane lining the lateral and posterior walls of the nasopharynx; so that the occurrence of adenocarcinoma also is to be expected.

In the series herein reported, 84 per cent of the growths were some form of epidermoid carcinoma, with malignant tumors of the salivary glands (adenocarcinoma) comprising 3 per cent. Fifty-six of the epidermoid carcinomas (77 per cent) were transitional cell carcinoma [12]

12. Quick, D., and Cutler, M.: Transitional Cell Epidermoid Carcinoma, Surg., Gynec. & Obst. **45**:320, 1927.

(42 cases) or lymphoepithelioma (14 cases). Highly differentiated squamous cell carcinomas (5 cases) and spindle cell carcinomas [13] (4 cases) made up 12 per cent. Within the past year, 2 histologically unusual neoplasms of the nasopharynx have been seen at the Memorial Hospital—1 a myxosarcoma and the other a notochordal tumor.

In the published reports, there are considerable disagreement as to the histologic terminology and a wide disproportion of percentages of groups in the classification of histologic types. While it is generally agreed that epidermoid carcinomas make up a fairly large proportion of all nasopharyngeal tumors, the designation of the other groups is rather confusing in its varied terminology, since many authors include various vaguely classified forms of sarcoma and endothelioma. One of the most disputed questions is the justification of the term lymphoepithelioma. Some observers [14] believe that there is no justification for the term, while others take an opposite view.[15]

TABLE 1.—*Histologic Types of Carcinoma of the Nasopharynx in a Series of 87 Cases Observed at Memorial Hospital*

A. Epidermoid carcinoma		73
(*a*) Squamous cell carcinoma	5	
(*b*) Transitional cell carcinoma, including the anaplastic varieties	42	
(*c*) Lymphoepithelioma	14	
(*d*) Spindle cell carcinoma	4	
B. Lymphosarcoma		11
C. Malignant tumor of the salivary gland		3
Total		87

During the preparation of this report, F. W. Stewart, pathologist at the Memorial Hospital, reviewed all the histologic slides in the present series and in a personal communication expressed the following opinion:

Within the broad group of epithelial tumors of the nasopharynx, certain subdivisions exist as more or less definite entities. As employed in the laboratories at the Memorial Hospital, the term "squamous carcinoma" applies solely to tumors which show definite keratinizing tendencies; otherwise the term "epidermoid carcinoma" suffices. The term "transitional cell carcinoma" is used to define more

13. Martin, H. E., and Stewart, F. W.: Spindle-Cell Epidermoid Carcinoma, Am. J. Cancer **24**:273, 1935.

14. Harvey, W. F.; Dawson, E. K., and Innes, J. R. M.: Debatable Tumors in Human and Animal Pathology: Lymphoepithelioma, Edinburgh M. J. **44**:549, 1937. Jovin, J.: Les lympho-épithéliomes du pharynx, Ann. d. mal. de l'oreille, du larynx **45**:729, 1926; Lymphoépithéliome et épithélioma à cellules de transition, Bull. Assoc. franç. p. l'étude du cancer **28**:117, 1939.

15. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928. Regaud, C., cited by Reverchon and Coutard: Lympho-épithéliome de l'hypopharynx traité par la roentgen thérapie, Bull. et mém. Soc. franç. d'oto-rhino-laryng. **34**:209, 1921. Schmincke, A.: Ueber lympho-epitheliale Geschwülste, Beitr. z. path. Anat. u. z. allg. Path. **68**:161, 1921.

orderly types of nonkeratinizing epidermoid carcinoma in which the picture suggests duplication of the intermediate layer of a stratified epithelium. In spite of suggestions that these tumors arise from transitional epithelium of gland ducts, no proof of such origin exists, nor is there proof that they always remain incapable of keratinization. Either a squamous or a transitional cell carcinoma may occur in more anaplastic form, in which case the term "anaplastic epidermoid carcinoma" is employed.

"Lymphoepithelioma" is a term which has enjoyed much recent popularity. It dates from the papers of Regaud and Schmincke and indirectly goes back to the work of Jolly, Stöhr and Retterer. Its significance is wholly dependent, so far as the morphology is concerned, on one's interpretation of the origin of the reticulum of the nasopharyngeal lymphoid tissue and the interrelation between epithelium and lymphocytes. It is certain that many pathologists were too ready to accept certain tumors as lymphoepitheliomas which previously would have been classed with lymphosarcomas. In reviewing a number of such cases within the past year, I have in several instances reclassified tumors regarded as lymphoepitheliomas and returned to the old designation of lymphosarcoma. These were tumors of the so-called Schmincke type. The question is not yet settled, and the whole subject remains in an unsatisfactory state.

SYMPTOMS, MORBID ANATOMY AND CLINICAL COURSE

It is characteristic of this disease that the primary lesion in the nasopharynx causes relatively few if any symptoms in the early stages. This phenomenon can be explained readily by a consideration of the local anatomy.

The nasopharynx is a cavity with rigid walls, which, as a part of the respiratory tract, is much wider than the nasal cavities in front or the glottis below. Obstruction to breathing will not occur, therefore, except in the later stages, when the tumor has become large and bulky. No other function can be disturbed early in the course of the disease unless the growth arises near or in the orifice of the eustachian tube, so as to obstruct the opening and cause partial deafness. The most common site of origin, however, is the posterior wall definitely to one side of the midline or in one of the fossae of Rosenmüller.[16] In the latter areas the tumor must reach a diameter of 2 to 2.5 cm.—or even more—before it can cause either occlusion of the eustachian tube or obstruction to respiration. Since this area is not particularly sensitive to pain, it follows that in growths of the posterior wall local symptoms are characteristically

16. One of us (M.) has observed a case in which the posterior and lateral walls of the nasopharynx, as well as the upper surface of the soft palate, were the site of multiple papillomatous lesions covering at least half of the surface. There was marked associated ozena. The histologic diagnosis was papillary squamous cell carcinoma grade I. Treatment by radiation according to our usual technic resulted in complete primary regression followed by a recurrence during the second year of two or three warty papillomatous lesions on the upper surface of the soft palate. These were treated by actual cautery, and the patient has now remained well for almost five years.

absent, and in many instances it is not until cervical nodes appear that the patient suspects anything amiss. Since the growths in the nasopharynx tend to be highly anaplastic, they metastasize comparatively early. Such metastasis was the most common single first symptom (about 50 per cent) in our series of cases.

Initial Symptoms.—In the order of their frequency the first symptoms complained of in the series herein reported were cervical metastases, nasal obstruction or discharge, headache or local pain, and defective hearing or pain in the ear. Other miscellaneous complaints consist of symptoms vaguely referable to the throat or pharynx. Even though they are not the most frequent, it seems best to begin a discussion of symptoms by describing those produced by the primary lesion.

Nasal Obstruction, Bleeding or Discharge.—First symptoms referable to the nose occurred in a little less than one third of our cases. The most common of these was nasal obstruction. Only slightly less often the first complaint was of nasal discharge, often bloody. It is obvious that before a posteriorly situated nasopharyngeal growth can produce obstruction, it must be of sufficient size to extend forward and partially block the posterior nares. In the majority of such cases, cervical metastases, unsuspected by the patient, also are found. When the growth arises on the lateral wall, unilateral deafness should occur before nasal obstruction. Hemorrhage can occur when any fungating fragile tumor is traumatized in an attempt to clear the nasal passages by blowing the nose. Nasal discharge or bleeding as the secondary or late symptom occurred in over half of the cases in this series.

Unilateral Deafness.—Symptoms referable to the ear occurred first in about 10 per cent of all cases and usually consisted of persistent progressive unilateral deafness and occasionally of pain or discomfort in the ear. As a later symptom or as a complication following treatment by irradiation, disturbances in hearing are fairly common. These usually occur with growths which arise on the lateral walls so as to block the eustachian opening and result in the imbalance of air pressure in the middle ear. Deafness may later occur as a result of intracranial extension and pressure on the auditory nerve, as well as on other cranial nerves. In such a case, one type of deafness is often superimposed on the other so gradually that only by testing for bone conduction can one determine whether or not the eighth nerve is affected.

Symptoms of Intracranial and Intraorbital Extension.—One of the most characteristic features of nasopharyngeal cancer is its tendency toward direct extension intracranially and to the orbit. In addition to localized pain, the symptoms of such extension are progressive unilateral paralysis of the cranial nerves in about the following order: sixth, third, fourth, fifth, seventh and second, with occasional involvement of the

others. The first nerve arises too far anteriorly to be affected, and in any case a unilateral disability would be difficult to prove. In our series, after paralysis of the group mentioned, we have frequently observed involvement of the eighth, ninth, tenth, eleventh and twelfth branches. As might be expected from a gradually advancing tumor, the disability gradually extends from the sixth upward and downward in the numbered scale and can usually be demonstrated first as a slight disability in each branch, which goes on to complete paralysis.

The route by which the growth progresses from the nasopharynx intracranially is readily explained by a consideration of the local morbid

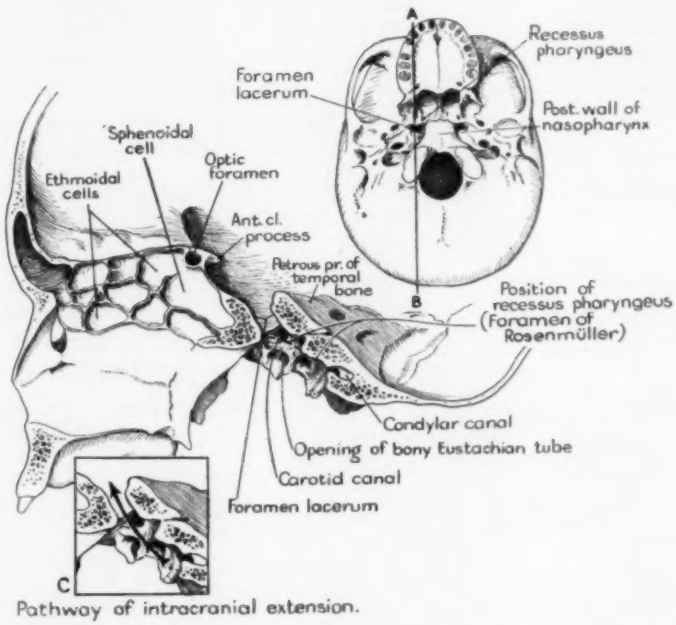

Fig. 2.—The fossa of Rosenmüller (recessus pharyngis) in its upper portion lies immediately over the foramen lacerum, which gives direct access for the intracranial extension of nasopharyngeal cancer. The large diagram is a sagittal section along the line *A-B* through the foramen lacerum, illustrating the juxtaposition of the lateral wall of the nasopharynx, the foramen lacerum and the cranial cavity. Insert *C* demonstrates the direct pathway of this extension.

anatomy of the disease (fig. 2). Although the growth may not arise directly in it, one of the fossae of Rosenmüller is practically always invaded fairly early in its course. These fossae of Rosenmüller lie directly under the mesial portion of the foramen lacerum. Because of the juxtaposition of these structures, cancer may extend by direct continuity of soft tissues from the nasopharynx through the foramen lacerum and into the cranium, a distance of about 1 cm., without the erosion of bone.

The foramen lacerum is filled partly by fibrocartilage, over the upper surface of which the internal carotid artery with its accompanying sympathetic plexus enters the foramen from the carotid canal, turning upward to enter the cranium. The foramen lacerum also transmits the superior petrosal nerve and occasionally a branch of the ascending pharyngeal artery.

In the later stages of intracranial extension, when several cranial nerves are paralyzed, it is often possible to demonstrate roentgenographically an erosion of the bony floor of the middle fossa. For this reason some investigators (Furstenberg,[17] C. L. Martin,[18] Ch'eng [19] and others) have attributed this complication to a unique capacity of nasopharyngeal cancer to erode the bone of the base of the skull. Needles [5a] reported that in about 60 per cent of the cases which he studied roentgenographically there was evidence of local pathologic changes in the bones adjacent to the nasopharynx, although not all of these changes consisted of definite erosion of bone.[20] C. L. Martin [18] stated that in about one third of all cases the sphenoid bone is invaded by cancer and in his report included excellent roentgenograms demonstrating this phenomenon. It seems to us that direct invasion by bone erosion is not *ab initio* the cause of intracranial extension of the disease. The local anatomy of the region, described in the preceding paragraph, permits direct intracranial extension of cancer through the foramen lacerum into the extradural space without the necessity of bone erosion. In a recent case in which there has been progressive involvement of the cranial nerves from the second to the twelfth with invasion of the orbit and marked exophthalmos for over five months, we have been unable to demonstrate any erosion of the bone of the base of the skull after repeated roentgenographic examinations of this region.

It has been repeatedly noted at autopsies that the intracranial extension of the growth tends to remain extradural. The dura forms the internal periosteum of the skull, and the growth advancing through the foramen lacerum will lie extradurally and therefore subperiosteally, and the internal table of the skull is therefore deprived of the protection of its growth-resistant periosteum. For this reason, once the disease has extended through the foramen lacerum the bone surrounding the foramen

17. Furstenberg, A. C.: Malignant Neoplasms of the Nasopharynx, Surg., Gynec. & Obst. **66**:400, 1938.

18. Martin, C. L.: Complications Produced by Malignant Tumors of the Nasopharynx, Am. J. Roentgenol. **41**:377, 1939.

19. Ch'eng, Y.: Lymphoepithelioma of the Nasopharynx with Involvement of the Nervous System, Chinese M. J. **49**:1075, 1935.

20. In our experience, the presence of an infected growth, often heavily irradiated, almost invariably causes some roentgenographically demonstrable changes in adjacent or underlying bone, which in most cases do not represent erosion by cancer.

and the floor of the middle fossa is attacked both from without and from within, and erosion of bone (if it occurs) is the result rather than the cause of intracranial extension.

The frequent early paralysis of the sixth nerve can also be explained on an anatomic basis (fig. 3). The nerve passes forward between the posterior clinoid process and the tip of the petrous portion of the temporal bone and therefore in its course passes directly over the foramen lacerum and nearer to it than any other cranial nerve. It is obvious, therefore, that this nerve should be the first involved by the extension of cancer through the foramen lacerum. The third, fourth and fifth cranial nerves lie next in proximity, just above the sixth, and are naturally involved next in frequency. When the disease extends forward into the

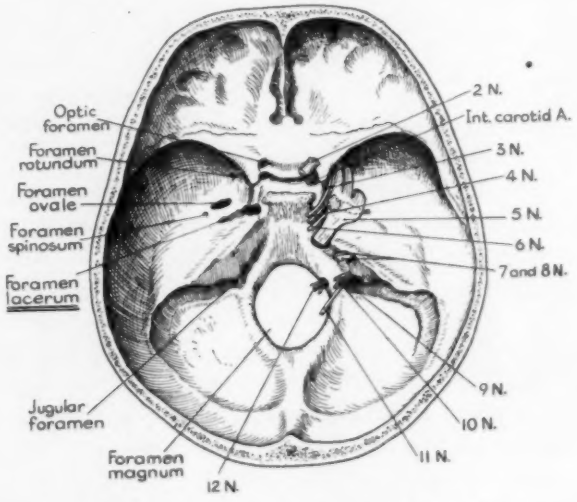

Fig. 3.—Diagrammatic representation of the base of the skull internally, showing the juxtaposition of the foramen lacerum and the third, fourth, fifth and sixth cranial nerves. Nasopharyngeal cancer extending intracranially through the foramen lacerum comes in contact first with the sixth cranial nerve. The growth may then spread forward through the superior orbital fissure into the orbit or backward into the posterior fossa to involve all the cranial nerves.

orbit there is pressure on the second nerve. The branches from the seventh to the twelfth, which lie further posteriorly, are involved only in the later stages of intracranial extension.

Intraorbital extension of the growth occurs by way of the foramen lacerum, the carotid groove and the superior orbital fissure. The tumor in advancing through the foramen lacerum comes in contact with the internal carotid artery, which, as it turns forward within the skull, lies in the carotid groove, and the growth advancing along this vessel and the groove approaches the superior orbital fissure, through which it

enters the orbit along with the several vessels and nerves. If paralysis of the second, third and fourth nerves has not already occurred from the growth expanding at the foramen lacerum, it obviously must occur when the tumor invades the superior orbital fissure, through which these nerves enter the orbit.

With invasion of the orbit, there are exophthalmos, ophthalmoplegia and eventually blindness. Schlivek [21] reported that about half of his 38 patients presented ocular symptoms. In a few of our cases, the patient stated that exophthalmos was the first symptom, which is evidence of the asymptomatic early course of the disease. Eventually, invasion of the orbit occurred in about 4 per cent of our series, accompanied by exophthalmos and ophthalmoplegia and by gradually increasing local pain, later diffused to the whole cranium of the affected side. In addition, intracranial extension is usually accompanied by a variety of symptoms, such as diplopia, headache and vomiting.

Origin Within or Extension Through the Eustachian Tube.—The growth may arise within or possibly extend through the eustachian tube, so as to be present both in the nasopharynx and in the ear, by perforation of the tympanic membrane. This phenomenon has occurred in several of our cases, usually as a fairly early symptom. In a recent case, the patient complained of pain in and discharge from the ear. Examination revealed a granular ulcerated lesion in the external auditory meatus and also a fungating tumor of the lateral pharyngeal wall on the same side. Biopsies disclosed adenocarcinoma. Treatment by fractionated roentgen irradiation directed through the opposite cheek and to the ear along the course of the eustachian tube resulted in complete regression, and the patient has remained well and free of disease for four years.

Furstenberg [17] reported a case of cancer of the eustachian tube with diagnosis made only at autopsy; despite the fact that the patient had cervical metastases and extension to the base of the skull, repeated biopsies of tissue from the nasopharynx gave negative results.

Cervical Metastases.—In the present series, cervical nodes were involved in 77 per cent of the determinate cases on admission, and while the patients were under observation metastases developed in an additional 4 per cent, making a total of 81 per cent in which they occurred at some time during the course of the disease. As has been previously mentioned, cervical lymphadenopathy was the first or only complaint on admission in about 50 per cent of the whole group. When present on admission, the nodes were between 3 and 8 cm. in diameter, and their duration, from the patients' statements, averaged about ten months. Not infrequently a patient with a node 3 to 5 cm. in diameter stated that it had suddenly

21. Schlivek, K.: Ocular Manifestations of Malignant Nasopharyngeal Tumors: Report of Cases, Arch. Ophth. **17**:1055 (June) 1937.

appeared within the preceding week or ten days when, obviously, judging from its size it must have been present for several weeks. In other cases, patients complaining only of local symptoms in the nasopharynx were found to have readily palpable enlarged cervical nodes of which they were not aware.

The presence of metastases on admission is one of the most important factors in prognosis, as will be discussed later. Cappell [22] gave cervical swelling as the initial symptom in only 20 per cent of his series, although the symptom was present in 60 per cent when the diagnosis was definitely established. Woltman [23] stated that metastasis was not common in his series, but Salinger and Pearlman [24] reported that 80 per cent of their patients showed metastases on admission, and Quick [25] reported 73 per cent in his series.

The lymph nodes most frequently involved are those of the upper deep cervical group lying just below the tip of the mastoid under the upper end of the sternomastoid muscle. Next in frequency are those situated just over the carotid bulb. After invasion of these upper deep cervical nodes, metastasis extends to the lower portions of the neck with more frequent dissemination to the posterior chain in the posterior triangle than occurs in cancer of the oral cavity itself. The submaxillary lymph nodes are rarely the initial site of metastasis, but in 1 case these and the submental nodes became involved after control of bilateral metastases in the upper deep cervical region. Widespread bilateral cervical lymphadenopathy is characteristic of the later stages of the disease.

There may be involvement of the lateral retropharyngeal lymph nodes which lie superficially on the prevertebral fascia a little above the level of the superior pole of the palatine tonsil. (According to anatomists, there are also the so-called median retropharyngeal nodes, which occur at a lower level near the midline.) Although these nodes lie closer to the nasopharynx than any others, they are not often involved at all and, if they are affected, are seldom the first. Metastatic involvement of the lateral retropharyngeal lymph nodes is evidenced by bulging of the posterior pharyngeal wall in this region a little above the upper pole of the tonsil, and it can easily be mistaken for a local direct extension of the primary lesion. It is unfortunate that in the present series specific observations regarding the invasion of this group of lymph nodes were not made in all cases.

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22. Cappell, D. F.: The Pathology of Nasopharyngeal Tumors, J. Laryng. & Otol. **53**:558, 1938.

23. Woltman, H. W.: Malignant Tumors of the Nasopharynx with Involvement of the Nervous System, Arch. Neurol. & Psychiat. **8**:412 (Oct.) 1922.

24. Salinger, S., and Pearlman, S. J.: Malignant Tumors of the Epipharynx, Arch. Otolaryng. **23**:149 (Feb.) 1936.

25. Quick, D.: Treatment of Malignant Growths of the Nasal Accessory Sinuses and Nasopharynx, Radiology **14**:191, 1930.

Systemic metastases are more frequent with cancer of the nasopharynx than with most other growths of the upper respiratory and alimentary tracts. Dissemination below the clavicle was present in 30 cases (34 per cent) of the present series on admission. The most frequent form of generalized metastasis was skeletal (pelvis, spine, long bones and ribs). The liver and the lungs are next in frequency, and these viscera tend to be involved in the later stages of all cases in which the disease is uncontrolled.

Late Clinical Course and Immediate Cause of Death.—When the disease is uncontrolled there are usually eventual intracranial and orbital extension and wide dissemination below the clavicle. The neurologic symptoms of intracranial extension have been described. With widespread involvement of the cervical lymph nodes, Horner's syndrome (enophthalmos, narrowing of the palpebral fissure, constriction of the pupil and anhidrosis) may develop. The early development of this syndrome in some cases can be explained by local extension of the disease along the internal carotid artery and its accompanying sympathetic plexus in the foramen lacerum. Diplopia, headache, dizziness, nausea and vomiting often accompany the later stages of intracranial extension.

Generalized dissemination is often heralded by pain in the back or in an extremity, indicating metastasis to bone. If such foci of the disease are not held in check by irradiation, pathologic fractures may occur. Visceral metastases are accompanied by the usual cachexia of widely disseminated cancer.

Death from nasopharyngeal cancer is almost always the result of widespread dissemination or intracranial extension of the disease rather than of the local effects of the growth in the nasopharynx. If the disease was not controlled, the average length of life in our series was about fourteen months. Other investigators have reported average survivals varying from ten months (Needles [5a]) to thirty-four months (New [26]).

DIAGNOSIS

There is probably no other form of cancer in which the true anatomic diagnosis is more often delayed or missed entirely than in cancer of the nasopharynx. As has been previously mentioned, this fault may be partly explained by the fact that in only about one half of all cases are there any early local symptoms from the primary lesion. In our series, 46 per cent of the patients had received previous surgical

26. New, G. B.: (*a*) Syndrome of Malignant Tumors of the Nasopharynx, J. A. M. A. **79**:10 (July 1) 1922; (*b*) Treatment of Malignant Tumors of the Pharynx and Nasopharynx, Surg., Gynec. & Obst. **40**:177, 1925; (*c*) Highly Malignant Tumors of the Nasopharynx and Pharynx, Tr. Am. Acad. Ophth. **36**:39, 1931.

treatment to relieve the symptoms without a correct diagnosis, and in only 30 per cent of the cases was the malignant nature of the condition suspected by the referring physician. There was an average delay of about ten months between the onset of symptoms and the establishment of a correct diagnosis.

Except for cancer, the nasopharynx is a relatively unimportant structure from the standpoint of disease, and it is therefore usually neglected in the course of an average physical examination. This region can be visualized only by difficult technical procedures, the most commonly used, although by no means the most efficient, being an endoscope. (The forms suitable for this region are variously denoted by such terms as nasopharyngoscope and antroscope.) Too much reliance should not be placed on the findings by these instruments alone, since,

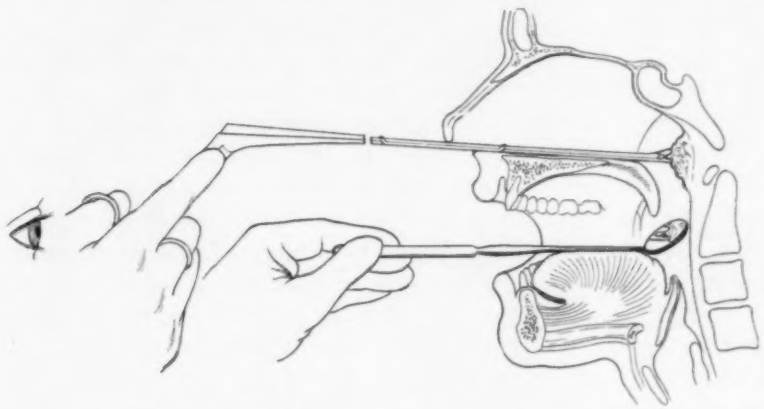

Fig. 4.—All parts of the nasopharynx are best examined by a mirror inserted through the mouth into the oropharynx. A biopsy specimen from this region may be removed through the nasal cavity, the operator being guided either visually by the mirror or digitally by the palpating finger.

because of their rigid systems of lenses, only a limited portion of the nasopharyngeal walls can be visualized. The most efficient method of examining the nasopharynx is by using a throat mirror in the pharynx while depressing the tongue and retracting forward the free edge of the soft palate. By this means, all parts of the nasopharynx are brought into clear view (fig. 4).

Even in the adult there is practically always some irregularity in the contour of the mucosa on the posterior wall of the nasopharynx, due to only partial atrophy of the lymphoid tissue of the pharyngeal tonsil. The primary lesion of cancer in these areas may consist of only a slight enlargement of one of these nodules. Such a cancerous nodule may or may not be ulcerated.

The fossae of Rosenmüller in the normal healthy adult may be rather broad, with smooth linings, or they may present irregularities. When one is searching for a primary lesion, any irregularity in these areas must be viewed with suspicion, especially if the two sides appear asymmetric. The primary lesion of cancer may manifest itself in one of these fossae by a slight increase in irregularity or by partial obliteration of the groove. In such cases, the biopsy forceps must be inserted rather deeply into the most irregular portion in order that a representative biopsy specimen may be obtained. A careful examination of, and a biopsy of tissue from, any suspicious area are indicated in any case in which cancer of the nasopharynx might be considered as a possible diagnosis.

Removal of a Biopsy Specimen from the Nasopharynx.—It is our opinion that, as with all forms of cancer, histologic confirmation of the diagnosis of nasopharyngeal cancer is essential before aggressive treatment is instituted. A single negative biopsy report should not be considered conclusive in the presence of a clinically positive tumor in the nasopharynx. In many instances of irregular granular tumors in the nasopharynx between 1 and 2 cm. in diameter, we have removed large portions successively and obtained a positive histologic diagnosis only after repeated attempts. In 1 instance, the biopsies gave negative results until the seventh specimen was removed and a diagnosis of anaplastic carcinoma was made. In our experience, in no other portion of the upper respiratory and alimentary tracts is cancer so apt to be overgrown by hypertrophic lymphoid or granulation tissue as in the nasopharynx.

A straight biopsy forceps inserted directly backward through the nasal cavity or a curved forceps inserted through the open mouth may be used to remove a specimen from a nasopharyngeal tumor (fig. 4). The manipulations should in either instance be guided visually by a mirror or digitally by a finger. We see no practical advantage in giving radiation treatment before taking the biopsy specimen, as recommended by Harmer.[27]

Cervical Lymphadenopathy.—As has been previously noted, cervical adenopathy is the first and only symptom of nasopharyngeal cancer in about 50 per cent of cases. In 70 per cent of the cases referred to us the primary lesion in the nasopharynx had not been suspected by either the patient or the referring physician. As we have postulated previously, it is highly probable that the majority of patients with nasopharyngeal cancer at large succumb to the disease without an anatomic diagnosis as to the location of the primary lesion.

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27. Harmer, D.: The Relative Value of Radiotherapy in the Treatment of Cancers of the Upper Air Passages, Lancet **2**:1057, 1931.

When the examining physician does not suspect the true character of the enlarged cervical node with nasopharyngeal cancer, it is not unusual for him to incise the node under a mistaken diagnosis of abscess or to excise a cervical node for biopsy. Some such procedures had been carried out in about 20 per cent of the cases in our series before admission. In 10 per cent, tonsillectomy had been performed under a mistaken diagnosis of chronic tonsillitis, which was believed to be the cause of supposed inflammatory lymphadenitis.

It cannot be too strongly emphasized that *cervical lymphadenopathy in the adult is almost always malignant and usually metastatic from a primary lesion in the upper respiratory or alimentary tract.* In the average patient who presents unexplained cervical lymphadenopathy, the nasopharynx is one of the most likely sites for a primary lesion of cancer. In such cases, the proper procedure is obviously to examine the oral cavity first and next the pharynx and the hypopharynx and then to proceed to the slightly more difficult and technical examination of the nasopharynx.

Aspiration biopsy [28] should be used to obtain histologic proof of the metastatic nature of clinically positive lymph nodes. Such information is essential if the clinical material is to be used for subsequent statistical analysis.

Unilateral Deafness.—In about 10 per cent of all patients with cancer of the nasopharynx, unilateral deafness is the first symptom. It must be realized that, although the collection of cerumen in the external auditory meatus is the most common, it is nevertheless not the only cause of unilateral deafness. This symptom, when slowly progressive in the adult, should always suggest the possibility of nasopharyngeal cancer. In our series, although cervical lymphadenitis was the most common first complaint which the patient considered serious enough to warrant seeking medical advice, closer questioning often revealed that slight unilateral deafness was actually the initial symptom.

Confusion with Other Diseases.—Furstenberg [17] reported that 25 per cent of his patients had been subjected to some type of operation before the correct diagnosis was established after the lapse of fifteen months, and Needles [5a] reported that 33 per cent of his patients had submitted to unnecessary operations. If the first complaint is partial nasal obstruction or hemorrhage, a mistaken diagnosis of nasal polyps or so-called "persistent adenoids" is likely to be made. In over 10 per cent of our series there had been repeated excisions of nasal polyps or of adenoids. In only a few of these cases had the surgeon suspected the possibility of cancer and obtained a histologic examination of the excised frag-

28. Martin, H. E., and Ellis, E. B.: Aspiration Biopsy, Surg., Gynec. & Obst. **59**:578, 1934.

ments of tissue to prove the presence of a malignant growth. In 1 case mastoidectomy had been performed because of the mistaken diagnosis of mastoiditis based on unilateral deafness and the presence of cervical lymph nodes. In another case, in which there was local extension to the orbit, a supposed orbital tumor had been excised with no suspicion of the primary lesion in the nasopharynx.

There are relatively few benign diseases or tumors occurring in the nasopharynx. Adolescent nasopharyngeal fibroma might be confused with cancer, but this benign tumor usually occurs only in adolescent boys and is characteristically bulky, smooth and of longer duration than cancer. We have observed 1 recent case of an apparent neoplasm of the posterior pharyngeal wall which proved to be tuberculosis of a retropharyngeal lymph node.

GENERAL PRINCIPLES IN THE TREATMENT OF CANCER OF THE NASOPHARYNX

There are two separate and distinct therapeutic problems in nasopharyngeal cancer—the treatment, first, of the primary lesion and, second, of the cervical lymph nodes. In general, the plan of treatment should be based on a consideration of the several characteristic features of the disease.

For all practical purposes, the primary lesion in the nasopharynx must be considered anatomically unsuited to adequate surgical removal. The nasopharynx is comparatively inaccessible to surgical exposure; the area is extremely vascular, and the growths, of whatever size, are poorly delimited, so that the difficulties of surgical removal, even of the smaller and more favorable lesions, are considerable. Other obstacles to surgical treatment are the frequent early extension of the disease intracranially and to the orbit.

On the other hand, nasopharyngeal cancer is one of the most radiosensitive of all tumors of the upper respiratory and alimentary tracts and, furthermore, this region tolerates heavy doses of radiation more satisfactorily than certain adjacent areas, such as the hypopharynx.[29] Therefore, if the disease has not extended beyond the nasal cavity itself, the primary lesions should be amenable to control by irradiation in practically all cases. For these reasons, there can be little question that irradiation is preferable to surgical removal for the treatment of the primary lesion.

Since cervical metastases occur in about three fourths of all cases, their successful treatment is one of the most important determining factors in the prognosis of this disease. Because the involvement is so

29. Martin, H. E.: Treatment of Pharyngeal Cancer, Arch. Otolaryng. **27**:661 (June) 1938.

often bilateral and widespread from a highly anaplastic tumor, neck dissection is probably of little value. In any event, bilateral neck dissection could hardly be carried out coincident with the aggressive irradiation to the nasopharynx. So far as we know, there are practically no reported cures of metastasis from nasopharyngeal cancer by surgical treatment alone, and those alleged to have been obtained by a combination of surgical intervention and irradiation probably depend almost entirely on the postoperative irradiation for the successful control of highly radiosensitive tumors.

General Hygienic Measures.—If, as in most cases, the irradiation must be given not only to the nasopharynx but also to the neck, mild radiation sickness always results, accompanied by loss of appetite and taste, malaise, slight dysphagia, lessening of the intraoral secretions and swelling and edema of the nasal mucous membranes with some nasal obstruction. The patient may be made much more comfortable if he is hospitalized for at least the two or three weeks of active treatment and the period of the reaction.

Treatment of the Primary Lesion.—The irradiation of the nasopharynx is probably best carried out by a combination of external irradiation through the cheeks and intracavitary radiation by radium or radon. Occasionally supplementary treatment to the primary lesion may be given perorally through the palate. Although cancer-lethal doses of radiation can undoubtedly be delivered to the nasopharynx by either one of these methods alone, a large proportion of undesirable effects of either method can be obviated by a combination of the two in less than their respective required maximum doses when used singly.

The treatment for nasopharyngeal cancer at the Memorial Hospital is ordinarily begun by fractionated roentgen irradiation through the cheeks (200 kilovolts; 50 to 60 cm. from T. S. D. [target-skin distance]; a filter of 0.5 to 1 mm. of copper; circular ports 6 to 7 cm. in diameter), the skin portals being centered anteriorly just lateral to the nose and the beam pointed obliquely backward and mesially so as to cross fire in the nasopharynx (fig. 5 *A* and *B*). The ports of entry may also be placed laterally so as to approach the nasopharynx horizontally through the coronal plane, although the skin portals themselves are thus subjected to a cross fire, the cutaneous reaction thereby being unnecessarily increased. Moreover, the possible intracranial extension may not be so efficiently irradiated unless such lateral ports are large.

In any case, the centering of these portals and the shielding should be so designed as to avoid unnecessary irradiation to the eye and the tongue. In the average patient, a portal of 6 cm. in diameter will be found to overlap the orbit and the upper lip slightly, although occasionally one 7 cm. in diameter may be used. To protect the globe itself, a slightly

convex oval lead shield (2 by 3 cm.) should be placed over the eye. A larger lead shield (fig. 5 *A* and *C*) should be placed in the mouth over the dorsum of the tongue, so that the latter structure is also protected from the effect of the direct beam. In the average case, the angle of the incident beam will be about 30 to 35 degrees from the sagittal plane, depending somewhat on the exact position of the portal. The determina-

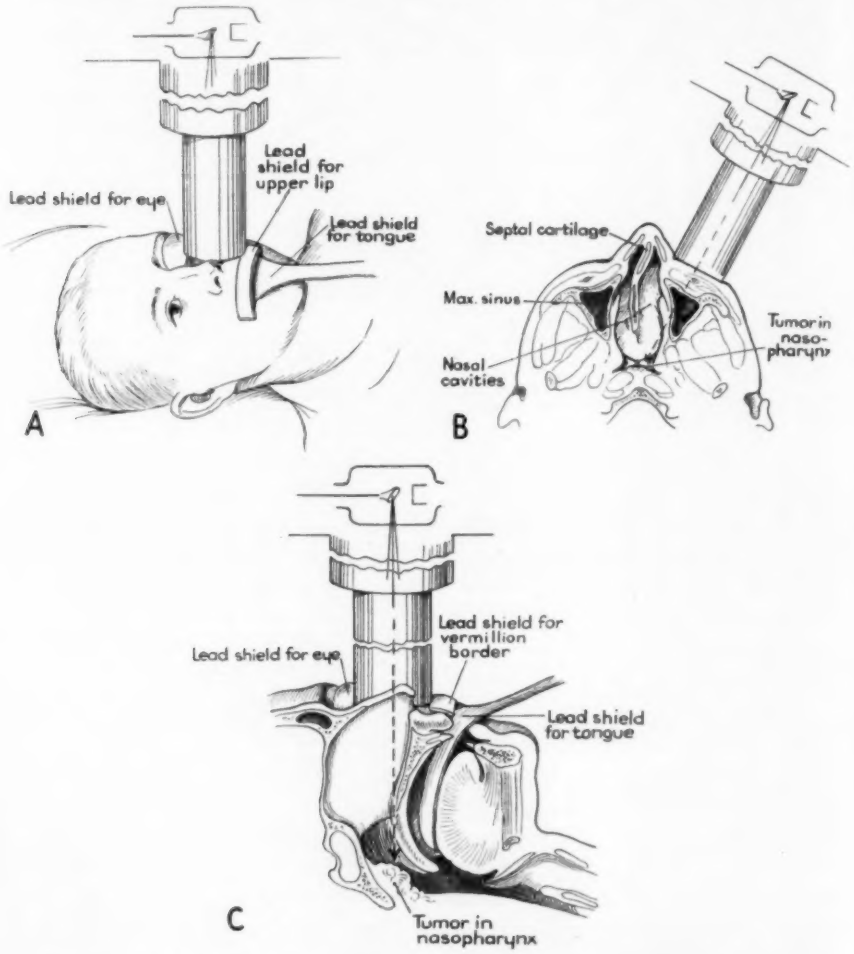

Fig. 5.—*A*, external irradiation of cancer of the nasopharynx through the cheek. One lead shield protects the eye; a second, the tongue, and a third, the vermilion border of the upper lip. *B*, external irradiation to the nasopharynx shown diagrammatically by a horizontal section. The beam is directed through the cheek inward and backward at an angle of about 35 degrees from the sagittal plane. *C*, external irradiation through the cheeks to the nasopharynx, showing the position of the lead shields on the tongue, the upper lip and the eye.

tion of these fine points in technic is somewhat empiric and should be based on personal observation by mirror of the anatomy of the nasopharynx and the position of the lesion.

Another minor point in the technic is the shielding of the vermilion border of the upper lip, which can be accomplished by a narrow strip of lead (fig. 5 *A*). If this area can be protected from the epidermicidal reaction, a great deal of later discomfort can be avoided.

With average-sized portals used as described, the daily dose should be 350 to 400 r applied to each cheek in rotation, with a total dose of about 3,000 to 3,500 r times 2 interrupted by the application of intracavitary radiation as will be described.

Peroral Roentgen Irradiation.—In order to spare the skin of the cheeks and the nasal cavity from too marked effects of irradiation, part of the external doses of radiation may be given perorally through the palate. This method of irradiation has already been described by one of us (M).[30] This variation in technic is not possible in some cases, because the patient is unable to open the mouth widely enough so that a peroral cylinder of sufficient size may be inserted and properly directed. In certain instances of a cooperative edentulous patient, this variation is distinctly worth while. A peroral cylinder at least 4 cm. in diameter with an oblique end should be used, and the direction of the beam carefully checked so that the lesion in the nasopharynx is accurately centered. The dose is variable, but with the portal size mentioned and a distance of 50 cm., a daily dose of about 400 r may be given in place of the usual dose to one of the cheek portals.

Intracavitary Irradiation.—By combining gamma radiation with roentgen radiation, the untoward effects of either of these agents used alone may be partially overcome. According to this plan, after the application of about 1,500 to 1,800 r to each cheek by roentgen irradiation, the treatment is interrupted and intracavitary radon applied, for a dose of about 1,000 millicurie hours, the skin in the meantime being given a four to five day rest from the roentgen irradiation. On the completion of this intracavitary dose, the roentgen irradiation is resumed until a total dose of about 3,000 r times 2 or more has been completed. The intracavitary radon capsule is then reintroduced for a total of 1,500 to 2,000 millicurie hours.

We formerly applied the intracavitary radon in a capsule tied into a rubber tube (fig. 6), by a technic already published.[31] To overcome the several deficiencies of this method, we have more recently employed

30. Martin, H. E.: Peroral X-Radiation in the Treatment of Intra-Oral Cancer, Radiology **28**:527, 1937.

31. Martin, J. E., and MacComb, W. S.: Protracted Irradiation by Radium, Am. J. Roentgenol. **37**:224, 1937.

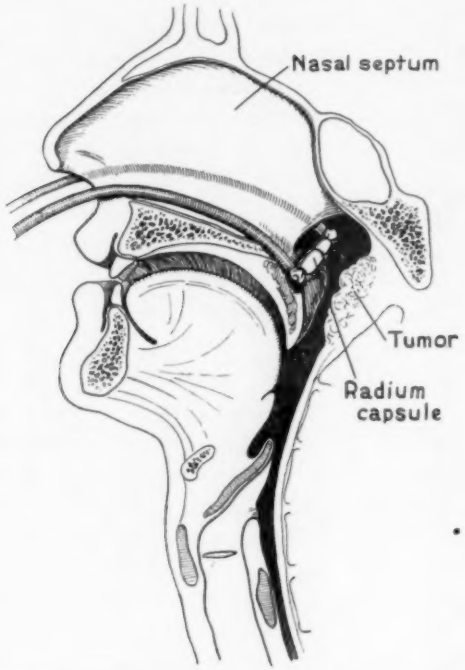

Fig. 6.—Intracavitary irradiation of cancer of the nasopharynx by a capsule of radium or radon tied in the center of a rubber catheter which is threaded into one nasal cavity and out the other. Although this method has the advantage of being simple and requiring no special apparatus, it is disadvantageous in that the capsule tends to lie on the upper surface of the soft palate rather than in contact with the tumor itself.

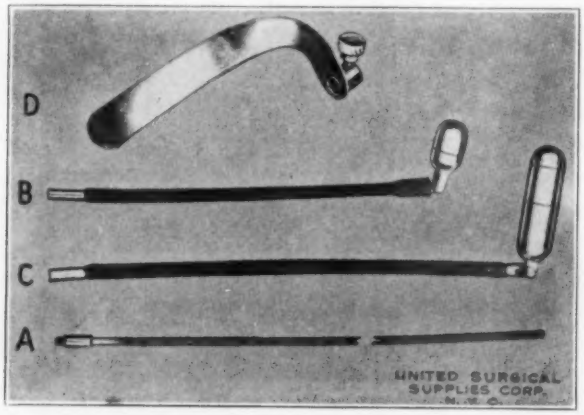

Fig. 7.—Nasopharyngeal radium applicator. *A*, steel shaft for inserting the applicator. *B* and *C*, radon capsules with attached shafts. *D*, immobilizing apparatus.

an improved applicator designed by one of us (B.).[32] The latter device consists of a metal capsule (wall thickness 1.75 mm. monel metal plus wall thickness of radon gold seed or radium needle), the halves of which are joined by a screw thread (fig. 7). This capsule, which is to contain the radiating source, is attached by a hinge to a flexible metal rod covered with rubber tubing. Either radon or radium element can be used as the radiating source, but in our clinic we have found radon seeds to be the most convenient, since the latter are always available in such

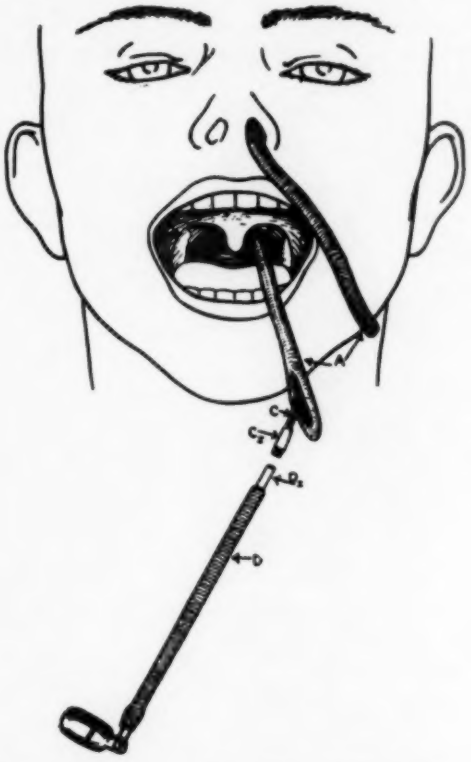

Fig. 8.—Insertion of nasopharyngeal radium applicator. A catheter (*A*) sheathing the flexible metal shaft (*C*) is threaded through the nasal cavity, into the pharynx and out of the mouth. The nasopharyngeal applicator (*D*) is then attached and drawn into the nasopharynx so that the shaft protrudes from the anterior naris.

quantities that the desired amount may be readily assembled without any particular preliminary preparation. To compensate for the decay of the radon, the applicator may be removed daily, if desired, and the

32. Blady, J. V.: New Instrument for Irradiation of Tumors of Nasopharynx with Radium or Radon, Am. J. Roentgenol. **40**:723, 1938.

decayed radon content replenished. According to our average technic, about 15 millicuries of radon is placed within the capsule and left in place ninety-five hours, giving a total dose of about 1,000 millicurie hours.

The device is inserted according to the following technic:

The selection of the nasal cavity through which the applicator is to be inserted depends on the position of the growth in the nasopharynx and the condition of the nasal passages. If the growth is definitely on one side, the corresponding

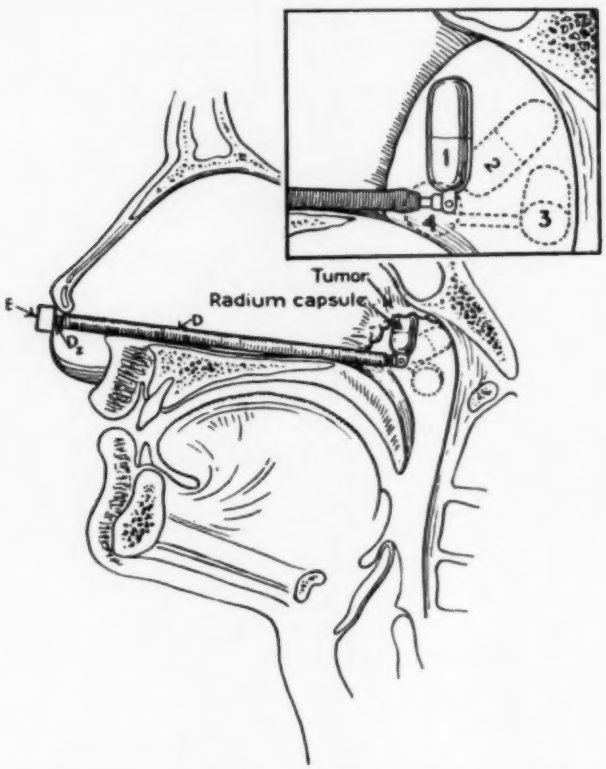

Fig. 9.—Nasopharyngeal applicator in place with a diagrammatic representation of the radon capsule in various positions.

nasal cavity should be used if possible, but if the growth is centrally placed on the posterior wall, the nasal passage of widest caliber will naturally be selected. For irradiation of growths in the roof of the nasopharynx, the capsule is placed erect so that it impinges or buries itself in the tumor (fig. 9). If the nasal cavity nearest the tumor is occluded or obstructed by an enlarged turbinate or deviated septum, the applicator can be inserted through the opposite nasal cavity. By oblique placing of the capsule in such a case, the radiation is properly delivered at the site of the tumor.

The selected nasal passage, pharyngeal and oral mucous membranes are anesthetized by topical application and spraying of a 10 per cent solution of cocaine hydrochloride. The flexible steel guide is sheathed with a no. 17 French catheter,

and the threaded end (fig. 8) of the guide is hidden in the aperture at the blunt end of the catheter. The latter is lubricated and inserted through the nasal cavity into the pharynx, and the blunt end is pulled out from the mouth. The threaded end of the guide is then pulled out of the catheter, and the applicator is attached to it. All metallic parts are pushed into the catheter, so that no irregular surfaces can traumatize the mucous membrane when the catheter is pulled out through the nose (fig. 8).

In this position, however, the capsule is not upright, but hangs down along the dorsum of the uvula. It can be readily brought into the desired position by rotating the end of the applicator shaft protruding from the anterior naris to the side corresponding with the nasal cavity being used. Then, by examining the nasopharynx with a mirror, the position of the capsule is checked so as to insure its being as nearly as possible in contact with the tumor. After the capsule has been properly placed, the immobilizing attachment (fig. 10) is fixed to the end of the applicator shaft protruding from the naris. Adhesive tape is placed securely from the free end of the attachment to the bridge of the nose. By spraying liquid

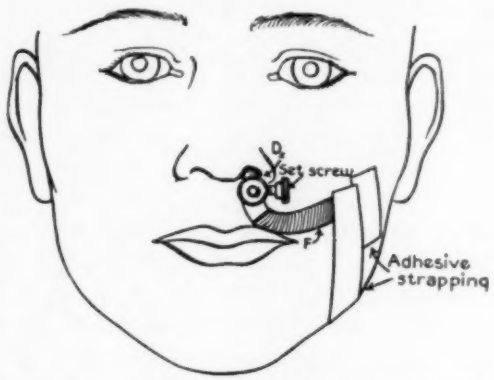

Fig. 10.—The applicator (*D*) held in place by the immobilizing attachment (*F*), which is fixed to the cheek by adhesive tape.

petrolatum into the nasal cavity and placing a wisp of cotton around the shaft at the anterior naris, contact trauma is largely prevented. The nasal cavity should be cleaned with a cotton applicator and liquid petrolatum as often as necessary.

To remove the applicator, the metallic end protruding from the nostril is detached, and by rotating the shaft the capsule is dislodged so that it hangs down into the pharynx or rests on the free edge of the soft palate. As the shaft is pushed into the nasal passage, the capsule presents itself in the oropharynx, where it can be grasped with the forceps and readily withdrawn.

Treatment of Metastases from Nasopharyngeal Cancer.—A detailed theoretic discussion of the treatment of cervical nodes does not come within the scope of this report. Briefly stated, it is our opinion that neck dissection is of little practical value in the control of cervical metastases from nasopharyngeal cancer, in which a highly malignant anaplastic radiosensitive growth tends to metastasize early and widely to both sides of the neck. The problem here, therefore, is more difficult than with the slowly growing and more highly differentiated tumors of

the lips, tongue or cheeks, in which the nodes more often tend to appear late in the course of the disease and to be unilateral and limited in extent.

In contrast to surgical intervention, irradiation is capable of permament control of a fair percentage of nasopharyngeal cervical metastases. It must be realized, however, that the injurious effects which limit the application of radiation are, in the general sense, similar to those of surgical intervention; that is, the dangers consist not only in local damage but in the deleterious constitutional effects of too aggressive treatment. In all cases, to be safe, the areas of either irradiation or surgical intervention must be limited; otherwise the effect on the general condition may be such as to cause a fatal termination.

It is the practice of some radiologists to irradiate the entire area of both sides of the neck on the theory that even though nodes are not palpable the dissemination is *probably* widespread. This practice would certainly be sound if cancer-lethal doses could be given safely to the entire potential lymph node–bearing area of the neck. It is our opinion, however, that the theoretic advantages of irradiation to the entire neck through large portals are more than nullified by the dangers and disadvantages of too widespread local and general deleterious effects. We have rarely observed the complete and permanent regression of bilateral *palpably involved* nodes after irradiation with portals covering the entire area of both sides of the neck from the mastoid to the clavicle. It has been our experience that if the widespread nature of the disease necessitates including the entire area of both sides of the neck, cancer-lethal doses usually produce such untoward sequelae that the patient, if he survives, is so uncomfortable and disabled as to render such treatment of little ultimate value. The fact that the patient may survive small doses of radiation given through large bilateral portals does not prove the efficacy of such treatment unless it can also be demonstrated that cancer was actually present when treatment was begun.

The foregoing comment is intended as a plea for the judicious limitation of the size of the portal to the requirements in the given case as evidenced by the extent and location of palpable or otherwise demonstrable involvement of cancer. With such limitation in area and volume of the tissues subjected to radiation, the tissue dose may be increased so as to produce a lethal effect on the tumor. The use of unnecessarily large portals will always prevent the safe administration of the larger doses which are usually required for the complete sterilization of the average cancer. The most successful treatments in our experience have been those in which a combination of fractionated roentgen irradiation and implantation of radon seeds was used. The latter supplement obviated the necessity of pushing the dose of roentgen radiation beyond its judicious limit.

Since the morbid anatomy of the metastases varies so widely, the actual technic as to portal size and daily and total dose of roentgen radiation and of supplementary interstitial radiation also varies widely. It is possible, therefore, to give only a few examples. If, as is often the case, there is bilateral involvement limited to the upper deep cervical nodes, two circular portals 7 to 8 cm. in diameter will often adequately cover all palpable evidence of the disease in the neck. In conjunction with the irradiation of the primary lesion in the nasopharynx, these two portals of the neck are irradiated daily, alternately, so that by the end of fifteen to twenty days a dose of 3,000 to 3,500 r times 2 has been given. It is usually preferable to stop short of the maximum dose and to implant (through the skin) radon gold seeds for a tissue dose of 6 to 7 skin erythema doses (S. E. D.) (table 2). As a

TABLE 2.—*Millicuries in Gold Seeds Required to Deliver Specified Doses to Masses of Various Diameters*

Skin Erythema Doses	Diameter of Mass—Centimeters											
	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	6.0	7.0	8.0
	Number of Millicuries											
5	1.0	2.5	4.0	7.5	10	12	14	17	20	27	35	45
6	1.2	3.0	4.8	9.0	12	14	17	20	24	32	42	54
7	1.4	3.5	5.6	10.0	14	17	20	24	28	38	49	63
8	1.6	4.0	6.4	12.0	16	19	23	27	32	43	56	72
9	1.8	4.5	7.2	14.0	18	22	26	31	36	49	63	81
10	2.0	5.0	8.0	15.0	20	24	29	34	40	54	70	90
11	2.2	5.5	8.8	17.0	22	26	32	37	44	59	77	99
12	2.4	6.0	9.6	18.0	24	29	35	41	48	65	84	108

variation, the roentgen irradiation to the neck may be interrupted for implantation of radon seeds and then, after an interval of five to six days, continued to complete the entire dose.

An example of the compulsory use of a larger portal is presented by 1 case of unilateral involvement in which there was a group of nodes in the upper deep cervical region about 7 cm. in diameter, with scattered isolated nodes about 1 to 5 cm. in diameter in the posterior triangle and under the lower end of the sternomastoid muscle. In this instance, in conjunction with the treatment of the primary lesion, an oval portal 18 by 13 cm. was applied to the affected side of the neck and irradiated with 150 r daily for a total of 5,000 r. At the completion of this dose, the skin was severely blistered over this whole area (fig. 11). The lower neck nodes completely regressed, but the large mass in the upper deep cervical region still persisted about 5 cm. in diameter. A total dose of 52 millicuries of seeds in fractions over the following eighteen days was then inserted into the residual mass, which subsequently also showed complete regression. This patient has remained well for over two years.

Treatment of Widely Disseminated Metastases.—Since nasopharyngeal cancer is practically always highly radiosensitive, the palliative treatment of widely disseminated metastases of this disease is distinctly worth while. As has been previously mentioned, bone metastases to the pelvis, spine or long bones are not uncommon. These lesions are almost always accompanied by persistent and often severe pain, which can frequently be completely relieved by moderate doses of external radiation. In such cases, it is advisable to localize the metastases exactly by roentgen examination or by palpation, so as to permit the accurate centering of a small portal, thereby treating the metastases by an adequate dose

Fig. 11.—G. P., a patient with nasopharyngeal cancer and unilateral metastases extending from just below the lobule of the ear to the clavicle, was given 150 r daily for a total dose of 5,000 r by fractionated irradiation to the neck through a portal 18 by 13 cm. (in addition to radiation of the primary lesion). After this, 52 millicuries of radon seeds was implanted in the residual mass in the upper part of the neck. The disease completely regressed. The skin healed, and the patient has remained well for over two years. Ordinarily, portals of this large size are not justified except when indicated by widespread extent of palpable metastases.

without too great a drain on the general tolerance. If such metastases cannot be accurately localized, it is obvious that moderately large portals should be used. It is often possible to relieve entirely the pain resulting from such deposits by the application of 1,000 to 1,500 r through a portal of only 6 to 8 cm. in diameter. By the same means, abdominal

metastases, in the liver or other viscera, may likewise often be rendered temporarily asymptomatic. It is not ordinarily profitable to irradiate widespread pulmonary metastases.

COMPLICATIONS OF TREATMENT

Effects of Irradiation on the General Condition.—It should be realized that the injurious effects of improperly administered radiation are often mistakenly considered limited to damage to the overlying skin or the local deep tissue. As a matter of fact, the most serious consequence, especially with the use of large portals, is the constitutional effect. In the treatment of nasopharyngeal cancer, the possibility of such systemic effects must always be kept in mind, since a relatively large volume of tissue must be irradiated through portals applied to both cheeks, to the nasopharynx and usually to both sides of the neck for cervical metastases. These effects consist of chronic radiation sickness due not entirely to the actual local discomfort or the local reaction but also to the large volume of tissue irradiated. The patient becomes listless, weak and toxic, loses weight and becomes cachectic. There may be also some damage to the base of the brain and partial derangement in function of the various glandular, vascular and neural structures of the neck. Such a constitutional effect cannot be entirely avoided if adequate radiation is given, but its degree may be considerably reduced by not using unnecessarily large portals.

Hospitalization for at least a week or ten days during the height of the reaction will do much to relieve the severity of the constitutional symptoms and to shorten convalescence. Aside from the care of the local reaction during the stay in the hospital, careful check must be kept on the caloric and vitamin content of the diet and the volume of fluid intake. Most patients appear to benefit from either peroral or parenteral administration of liver.

Local Reactions to Irradiation.—In most cases the treatment outlined in this report will produce a transitory epidermicidal reaction in the skin of the cheeks and neck, usually healing within fifteen to twenty days from inception. At first, the involved skin may be kept dry or lubricated by the application of liquid petrolatum. When the epidermis has become broken and partly denuded, the area should be kept covered by a bland ointment (boric acid ointment) spread on fine mesh gauze.

The mucosal reactions in the nasopharynx go on to a membranous mucositis, which usually involves the under surface of the soft palate as well as the nasopharynx and the nasal cavities. These areas should be kept clear of the viscid mucous discharges by frequent irrigations with an alkaline and saline solution (4 Gm. each of sodium chloride and sodium bicarbonate in 1,000 cc. of water). The nasal syphon is

useful for this irrigation. It consists of an irrigating can with a rubber tube and hard rubber nozzle to fit in one anterior naris with another nozzle and rubber tube in the opposite naris. The solution thus enters one nasal cavity and passes through the nasopharynx and out the other nasal cavity. Following such irrigations, any remaining crusts or clots of secretion may be removed with a forceps and the cavity sprayed with liquid petrolatum. When irradiation is given to the neck for cervical nodes, there is usually some mucositis in the pharynx, which should also be irrigated several times daily.

If the eyes have been properly protected by shields of lead during irradiation, no reaction should occur in the eyelids and conjunctiva, an important factor in promoting comfort and a shortened convalescence, as well as in reducing the incidence of late radiation cataract. No reaction should occur either in the tongue or in the vermilion border of the upper lip if these structures have been properly shielded in the manner already described.

Pain.—With nasopharyngeal cancer, pain ordinarily occurs only after local extension of the disease to the orbit or intracranial cavity or as the result of bone metastases. With properly administered treatment, it should not occur as the result of localized radionecrosis.

Although palliative irradiation is highly successful in alleviating the pain of bone metastases, it is of only moderate value for the relief of symptoms due to extension to the orbit, the base of the skull and the intracranial cavity. However, with such local extensions of the growth, an attempt at palliation should always be made by fractionated roentgen irradiation with reasonably small portals (5 to 8 cm. in diameter) properly centered over the involved area as shown by roentgenograms or neurologic examination. In cases of intracranial involvement, the administration of too large doses should be avoided, since thereby the symptoms will only be made more severe. We have not found the relatively large portals, including both the nasopharynx and the middle fossa of the skull, recommended by C. L. Martin,[18] to be of ultimate benefit in the cure, the prevention or the palliation of intracranial extension. For the later stages of uncontrolled nasopharyngeal cancer, maximum doses of opiates are required to temper the suffering.

Radionecrosis.—This complication in the soft tissues of the nasopharynx may result from too intense local irradiation, especially by the intracavitary method. It is particularly apt to occur following attempts at treatment of local recurrences. It should not result even with cancer-lethal doses if the radiation has been administered by a combination of submaximal doses of external and of intracavitary application. Radionecrosis in the nasopharynx is usually accompanied by persistent pain, often so severe as to require opiates. This complication is not always

fatal, at least not immediately, as is shown by several of our patients who have survived for several years without recurrence of the disease, although an area of bone 2 to 3 cm. in diameter is exposed in the posterior nasopharyngeal wall. As time goes on, although never completely comfortable, these patients usually finally attain a state of pathophilia consistent with a tolerable existence.

Recurrence After Treatment by Irradiation.—If the growth recurs in the nasopharynx after local irradiation as already described, the same dose cannot be repeated even after a long interval, since the local tissues never return to normal. In the average case, the patient will probably be better off if such local recurrences are left alone. Recurrent metastatic cancer in the neck may often be successfully treated by further irradiation, especially when the metastatic node occurs outside the previously irradiated area.

PROGNOSIS

Despite frequent gloomy reports in the literature (Hauser and Brownell,[33] Patterson,[34] Furstenberg,[17] Bazzana,[35] Needles,[8a] Simeoni,[36] Schlivek,[21] Worms and Carillon,[37] New [26]) nasopharyngeal cancer is by no means a hopeless disease if properly treated by aggressive irradiation. As will be shown later, under a report of end results, the prognosis is about the same as in cancer of the tongue even if all cases in all stages of the disease are counted.

Many factors influence the five year cure rate, as shown in table 3, and although our present series is not large enough to enable us to present conclusive evidence in this regard, the following analysis should be of interest.

Age.—From this analysis, it appears that the prognosis is least favorable in the young and in the very old and that patients between the ages of 50 and 59 have the best chance of cure. This variation of the prognosis with age can be partly explained by the extremely malignant character of nasopharyngeal cancer in infants, in children and in young adults. In these young subjects, especially in children, systemic metas-

33. Hauser, I. J., and Brownell, D. H.: Malignant Neoplasms of the Nasopharynx, J. A. M. A. **111**:2467 (Dec. 31) 1938.

34. Patterson, N.: Use of Diathermy Knife in Malignant Disease of Mouth, Nose and Pharynx with Analysis of One Hundred and Nine Cases, Lancet **2**:633 and 694, 1934.

35. Bazzana, E.: I tumori maligni del rinofaringe, Oto-rino-laring. ital. **6**:319, 1936.

36. Simeoni, C.: Sui tumori maligni del rinofaringe, Boll. d. mal. d. orecchio, d. gola, d. naso **55**:81, 1937.

37. Worms, G., and Carillon, R.: Tumeurs du rhinopharynx à propagation cranienne: De leur diagnostic précoce; importance de l'examen radiologique, Rev. d'oto-neuro-opht. **8**:161, 1930.

tases appear almost invariably, even though the result of the treatment of the primary lesion may be favorable. For the decade between 50 and 59, extremely malignant and unfavorable tumors make up a much smaller proportion of the whole group. For patients over 60, the low cure rate is undoubtedly due to a less robust general condition and a lack of tolerance for the complications which often accompany the treatment. The youngest patient to recover in this series was 13 (anaplastic

TABLE 3.—*Factors Influencing the Five Year Cure Rate in 87 Cases of Cancer of the Nasopharynx Observed at Memorial Hospital from 1930 to 1934*

	Total Number of Cases	Number of Five Year Cures	Percentage of Five Year Cures
Age in years:			
Below 40	32	7	22
40 to 49	21	5	24
50 to 59	19	6	32
Over 60	15	2	13
Sex:			
Males	68	15	22
Females	19	5	26
Metastases:			
None at any time	15	7	47
None on admission	18	7	39
Present on admission	62	13	21
Sometime during course	65	13	20
Developed after admission	3	0	0
Bilateral metastases	17	4	24
Position of cervical nodes involved:			
Upper part of neck, one side	31	4	13
Upper part of neck, both sides	17	3	17
Entire neck, one side	10	2	20
Carotid bulb, single node	7	4	57
Extension of the disease to the base of the skull:			
On admission	3	0	0
Developed after admission	28	2	7
Histologic classification:			
Lymphosarcoma	11	3	27
Malignant salivary gland tumor	3	0	0
Epidermoid carcinoma	73	17	23
Squamous carcinoma	5	1	20
Transitional cell carcinoma	42	10	24
Spindle cell carcinoma	4	0	0
Lymphoepithelioma	14	6	43

carcinoma of the nasopharynx with bilateral cervical metastases). We have not observed recovery from the disease in patients below that age. The oldest patient to recover in this series was 64.

Sex.—As in most forms of cancer of the upper respiratory and alimentary tracts, especially those treated by irradiation, the prognosis in the female is slightly better than in the male. This tendency is probably due to the fact that, in general, any form of cancer in the female tends to be more radiosensitive than its anatomic counterpart in the male.

Metastases.—The presence of metastases in nasopharyngeal cancer, as in most other forms of the disease, is one of the most important determining factors in the prognosis. As will be seen from table 3, the rate of cure among patients without metastases at any time was almost

50 per cent. If metastases were not present at the time of admission but developed later, the cure rate was almost 40 per cent. The presence of metastases on admission reduced the chances of cure to less than average. The presence of bilateral metastases, however, did not markedly alter the average prognosis.

Histology.—From the histologic standpoint, one noteworthy finding in an analysis of our cases is that the prognosis with lymphoepithelioma (42.9 per cent) is much better than that with any other form of the tumor or than the average (25 per cent). The cure rates of lymphosarcoma and transitional cell carcinoma are near the average for the whole group. Unusual pathologic varieties, such as salivary gland tumors and spindle cell carcinoma, carry a poor prognosis.

END RESULTS

In the literature, reports of cures in series of 10 or more patients range from almost zero (Furstenberg [17]) to 20 per cent (Cappell [22]). So far as we can find, there are no well attested reports of cures by methods other than irradiation. The best figures are those of Gerlings and den Hoed,[6] who reported 12 per cent five year survivals in 42 cases, and Cappell, who reported 2 five year survivals (20 per cent) in 10 cases. In many otherwise excellent papers [38] the end results are based on survivals for varying periods of less than five years or on only those patients who were traced, so that actual five year end result figures cannot be accurately determined.

The five year end results in the series herein reported are given in table 4, and in connection with this report some explanation of the method of statistical analysis is necessary. It has been the policy of the Head and Neck Clinic of the Memorial Hospital for many years to accept all ambulatory patients for treatment or palliation, without excluding any because of the advanced stage of the disease. If the patient is not ambulatory, either because of his cancer or because of any associated physical, social or economic condition, he must obviously be considered unacceptable for treatment and, therefore, logically unsuitable for statistical purposes. Since practically all patients with cancer of the head and neck are ambulatory, so far as the tumor is concerned, almost until the time of death, few patients are excluded for this reason. If the patient makes only one or two visits and disappears within a month ("clinic shoppers"), he is not included in our calculations. We

38. Bazzana.[35] Gerlings and den Hoed.[6] Needles.[5a] New.[26c] Salinger and Pearlman.[24] Wagers, A. J.: Sarcoma of Nasopharynx with Report of a Case, Laryngoscope **48**:302, 1938. Crowe, S. J., and Baylor, J. W.: Benign and Malignant Growths of the Nasopharynx and Their Treatment with Radium, Arch. Surg. **6**:429 (March) 1923. Heine, L. H.: Malignant Tumors of the Nasopharynx, Arch. Otolaryng. **22**:51 (July) 1935.

believe that such a policy for the collection of clinical material is the best which has yet been established for the calculation of true statistics as to the possibility of cure of the various anatomic forms of cancer as this disease exists at the present time.

The proof of cure is the positive knowledge that a patient has remained alive and well for five years. Every effort is made to follow all patients by periodic observation in the return clinic. If this is not possible during the full five years, positive proof must be obtained in the form of a written or oral statement by the patient, the family doctor or a relative that the patient has survived and is well at the end of five years.

TABLE 4.—*Five Year End Results in Cases of Cancer of the Nasopharynx Observed at Memorial Hospital from 1930 to 1934*

This series consists of the cases of all patients with histologically proved cancer of the nasopharynx, both early and advanced, admitted during the specified period. Only those patients are excluded who, for any reason, were unable to return for treatment, palliation and observation in the outpatient department, and those who were lost track of within the first month after no more than one or two visits ("clinic shoppers").

Total number of patients		87
Indeterminate group:		
Dead as a result of other causes and without recurrence	4	
Lost track of without recurrence	3	
Total number of indeterminate results		7
Determinate group:		
Total number minus those of indeterminate group		80
Failures:		
Dead as a result of cancer	60	
Lost track of with disease (probably dead)	0	
Living with recurrence	0	
Total number of failures in treatment		60
Successful results:		
Free from disease after five years or more		20
Net five year end results:		
Successful results divided by determinate group (20/80)		25 per cent

During this five year period following treatment, a number of uncontrollable factors will tend to modify the outcome in the group as a whole, so that we have felt it essential before calculating the net end results to subtract a so-called "indeterminate group"; that is, patients who die of other causes without recurrence and patients lost track of after being free of disease for at least one year. Forty-five is the average age of patients with cancer of the nasopharynx, and actuarial statistics reveal that deaths from causes other than cancer in the five year period after 45 is about 4 per cent. It is obvious that corrections for this factor in a given series of cases of nasopharyngeal cancer must, therefore, not grossly exceed such a figure. In regard to corrections for untraced patients, biometric statisticians are in agreement that at least 90 per cent of the whole group must be traced in order that the net figures may be accurate.

Of the 87 patients with nasopharyngeal cancer herein reported on, the results for 7 are considered indeterminate (4 died of other causes; 3 were untraced). There remains a determinate group of 80 patients, of whom 20 are living and well 5 years or more, giving a net cure rate of 25 per cent.

SUMMARY AND CONCLUSIONS

Cancer of the nasopharynx makes up about 2 per cent of all malignant growths of the head and neck.

Because of the absence in many cases of early local symptoms in the nasopharynx, the diagnosis is frequently delayed or missed entirely.

The disease is characterized by early metastasis to the cervical lymph nodes and extension intracranially and to the orbit.

The surgical inaccessibility of the primary lesion and its high radiosensitivity make irradiation the treatment of choice, and a combination of external and intracavitary irradiation gives the best results.

Cervical metastases from this rapidly growing, highly anaplastic tumor also are best treated by irradiation.

In the Head and Neck Clinic at the Memorial Hospital, 87 patients were treated from 1930 to 1934, inclusive, with a net five year cure rate of 25 per cent.

CONSERVATIVE TREATMENT OF SINUSITIS IN CHILDREN

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There is a definitely increasing recognition of the prevalence of nasal sinusitis in children. A variety of conditions formerly undiagnosed or called nasal catarrh are now known to be nasal sinus infection. When the family physician or the pediatrician keeps in mind the possibility of nasal sinus infection, the actual diagnosis is usually not difficult.

By the proper study and treatment of the sinuses the frequency and duration of colds in children, till now always discouraging to parents and physicians alike, can be reduced.

The ensuing remarks are not meant to apply so much to the cases in which the nature of the condition is more obvious by virtue of a purulent nasal discharge or to the acute sinus infections with high temperatures, prostration or signs of purulent invasion of the orbit or meninges. In such cases the local condition usually permits of no serious diagnostic error; the patient is fairly certain to receive the indicated active attention.

In this essay I wish to draw attention to the type of case in which the condition is not so readily recognized as sinusitis. Emphasis will be placed on diagnosis and conservative physiologic treatment.

DEVELOPMENT OF THE SINUSES

The following brief outline of the development of the sinuses will perhaps be sufficient for the purpose:

The cavities of the paranasal sinuses are formed by the absorption and evagination of the respective regional bones by the nasal mucous membrane. At birth the ethmoid sinuses are the only ones consistently present and occupying their permanent relative position. The frontal sinus is only potentially present, in that it is formed from the anterior part of the ethmoid system. The maxillary antrum is present as a mediolaterally flattened cell on the nasal side of the orbit. The sphenoid sinuses are usually undetectable.

When the child is about 6 years of age the frontal sinuses have progressed to a position which is partially in the vertical portion of the frontal bone. The antrum has moved laterally and downward to its

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permanent location under the orbit. The sphenoid sinus can be recognized as a sinus during the second year.

These phases in the development of the nasal sinuses represent the average but are far from invariable. Clinical and roentgen examination often reveal a stage of development (pneumatization) later or earlier than the average for the patient's age.

From the practical point of view a consideration of the foregoing outline indicates that in the early years only the ethmoid sinuses and to a lesser degree the maxillary antrum can share in the nasal inflammations. After this period all the nasal sinuses are subject to infections of clinical significance.

PHYSIOLOGY AND PATHOLOGY

In infants the sinuses exist only as outpocketings of the nasal mucosa. Thus, infections of the sinuses must of necessity coexist with infections of the nasal mucosa, and clear up when the nose clears up. As the child grows the size of the sinuses changes radically in proportion to the width of the respective ostiums. In this way there occurs a small opening to a relatively large sinus cavity. This disproportion makes it much harder for the sinus to empty itself and increases retention and stagnation within the cavity. The normal physiologic processes of the mucosa must suffer from the continued presence of undrained products.

Another feature of sinus structure which unfortunately furthers the retention of secretions is the location of the ostiums. In the antrums and sphenoid sinuses particularly, the openings by which the cells communicate with the nasal chambers are so inefficiently placed for drainage, so far above the level of the floor of the sinus cavity, that deep layers of irritating and toxic inflammatory products can accumulate. With the ostium situated above the fluid level of the sinus contents, unless there is a marked change from the erect posture of the head, the sinus cannot empty itself with any degree of efficiency, and again retention and stagnation result.

The remarkable capacity of the nasosinal ciliated epithelium to cleanse itself is not unlimited, and even in spite of its well known ability to work against gravity, there comes a time when the cilia must succumb, at least temporarily, to the overwhelming blanket of pathologic products.

Since the mucosal linings of the nasal sinuses and nasal cavity proper are merely continuations and ramifications of the same epithelium, a common cold involves the entire extent of the membrane. A common cold is thus a rhinitis and a sinusitis (rhinosinusitis). When the usual cold, running its course in the ordinary number of days, clears up, the sinusitis and the rhinitis resolve at the same time. This sinus involvement was part of the cold and not clinically sinusitis per se. It is only when some combination of factors causes the sinus part of the infection to persist

after the normal, so-called self-limited course of the cold has finished that there are the beginnings of a clinical sinusitis.

This persistence of the sinus part of the infection may be due to a variety of factors. On the part of the invading organism, the causative factor may be a special virulence such as occurs in the exanthems, in epidemic influenza or in streptococcic infections. On the part of the host, the general state of the patient's health and resistance are intangible considerations which have an undoubted bearing on the outcome. Natural morphologic features previously mentioned and local anatomic variations may hinder resolution when impediments to ventilation and drainage give rise to retention of discharges.

DIAGNOSIS

As in the case of any condition under a physician's consideration, the patient's history, previous and present, must be reviewed.

The first line of inquiry should be about the occurrence of colds. If the parent has not already volunteered the information, questions will invariably elicit the history that colds are frequent. In fact, the parents are discouraged because there seems to be so little they can do about diminishing the number of colds. They have usually been through most of the routine procedures, viz., injections of vaccine, the use of nasal tampons, vitamin therapy, administration of tonics and others.

Often the onset of the chain of complaints is found to be related to an attack of measles or scarlet fever. These diseases frequently have a specific complicating sinus infection which dies down without really completely clearing. Every succeeding cold causes a flare-up of greater or lesser intensity, and eventually the colds are more frequent and more protracted until the realization is reached that the matter is one not of colds per se but of recurring attacks of sinusitis.

The same sequence of colds of increasing frequency and duration needs no measles or scarlet fever to start it on its course, but these specific diseases can provide an excellent starter.

The commonest complaints presented for consideration are: (1) the frequent colds just described, (2) nasal obstruction and discharge, (3) some degree of low grade fever and (4) cough.

Nasal discharge points obviously to the region involved. This complaint has always been vaguely labeled nasal catarrh, with no insight into the actual physiologic and histologic anomaly. The discharge varies in amount and quality. It is usually some form of mucopus, although occasionally it is clear. The amount of actual pus in the mixture depends on the duration of the condition, the amount of tissue reaction and the nature of the infecting organism. It depends also on the amount of obstruction to drainage and the opportunities afforded for retention of discharges in the undrained cells, crannies and tissue crevices of the nose.

Secondary infections in an allergic nose usually produce large amounts of mucus in the discharge. Smears of the discharge properly stained (Wright's or Giemsa's stain) show a large number of eosinophils when an allergic factor is present. It is instructive to note the replacement of the eosinophils by neutrophils when infection supervenes and the return of the eosinophils when the subsiding infection leaves the underlying allergic status.

The low grade fever, when not associated with such a diagnostic lead as nasal discharge, has not until recently been given sufficient thought from the standpoint of nasal sinus infection.

The type of temperature that probably leads to the most diagnostic confusion is the low grade persistently elevated type which the examiner ascribes to the "cold," to the pharyngitis or to associated cervical lymphadenopathy. These findings are part of the same picture and should be considered collectively, not individually or symptomatically. As days and even weeks go by, the physician begins to worry about the temperature, its duration and its generally depressing effect on the child's status. The possibility of typhoid fever, tuberculosis, undulant fever and other such systemic infections is carefully ruled out by all available procedures.

The cough which so frequently accompanies the postnasal dripping of sinusitis is due partly to irritation and partly to infection of the lower tract by direct contiguous spread. The discharges find their way to the lower part of the pharynx, the laryngeal aditus and possibly into the larynx and lower air passages. The cough is often characterized by its occurrence in morning spasms on the child's arising. The change in posture when the patient gets out of bed brings a plug of discharge down from the nasopharynx, where it has accumulated during the relative inactivity of sleep. The discharge comes into contact with new areas of mucosa, unaccustomed to its presence, and the patient coughs reflexly or purposefully to get rid of the irritant. When the drip is thick and tenacious, efforts at dislodgment may provoke spells of gagging and even vomiting. This gagging and vomiting are rarely thought of by the laity as being due to nasal trouble. "Nervous stomach" is the common parental opinion.

With these complaints and a history as described, diagnostic consideration of the nasal sinuses should be given a prominent place. It is the purpose of this paper to emphasize the frequency and importance of nasal sinus infection in children so that the condition will be thought of more often. The actual diagnosis is usually not difficult. It can be confirmed by the steps outlined in the following paragraphs.

Rhinoscopic examination reveals some degree of congestion and diffuse engorgement of the nasal mucosa. Usually the condition is more pronounced over the middle turbinates. The airways will be seen to be

encroached on and narrowed by the boggy mucosa, little or no room being left for adequate drainage.

With the swollen middle turbinates tight against the lateral nasal wall, drainage from the anterior group of sinuses is impeded to a marked degree. It has been shown earlier that the commonest sinal involvement in children probably occurs in the maxillary antrums and in the ethmoid cells. It is the drainage from these sinuses that is blocked by the edema of the middle turbinates. Under such circumstances, discharge may not be obviously profuse and a common clue to the diagnosis may be lacking. The appearance of the tissues, however, gives adequate evidence of the regional pathologic condition. Careful, gentle topical shrinkage may reduce the swelling sufficiently to bring on some drainage. A displacement irrigation almost always brings out enough pathologic material to provide diagnostic evidence.[1]

Inspection of the oropharynx gives another lead to trouble higher up. Associated with postnasal discharge of any duration there is a secondary effect on the pharyngeal mucosa. Instead of the usual smooth, glistening normal appearance of the lining, there is presented a granular, pebbled appearance, due to irregular thickening of lymphoid tissue. It looks like a miniature, irregular layer of cobblestones with the elevations a deeper red than the intervening areas. The elevations may appear only in individual isolated spots, or they may be numerous enough to cover the entire pharyngeal wall.

On each lateral pharyngeal wall behind the posterior pillars of the tonsils may be seen a similar thickening in the form of a vertical band coming down from the nasopharynx and blending at about the level of the floor of the mouth with the more normal tissue. These lateral pharyngeal bands may be unilateral or bilateral. When unilateral the band points to the side on which the nasal sinus drainage is most profuse and gives evidence of the downward pathway of the irritating postnasal discharge.

In association with either of the mucosal reactions in the pharynx which were just mentioned, is usually seen the causative mucopurulent discharge. This discharge, as has already been stated, varies widely in color, in amount and in tenaciousness. It may be a colorless, clear, glairy mucus, and it may be a thick greenish to greenish brown material, more fluid and less tenacious. There are many gradations between these extremes.

On the other hand, there may be little or no visible discharge at the time of examination. Only the mucosal reaction to the acrid, irritating postnasal dripping is seen. The discharge may have been momentarily

1. Shambaugh, G. E., Jr.: Obscure Low Grade Chronic Infections of the Ethmoid Sinuses, J. A. M. A. **112**:1226 (April 1) 1939.

removed by hawking and expectorating. In such cases reexamination after an interval reveals the drip. In children large masses of adenoid tissue in the nasopharynx can mechanically obstruct ventilation and drainage. Such adenoid vegetations must be removed surgically if good results are to be expected of any type of treatment.

When no great amount of secretion is evident and other findings suggest sinusitis, a valuable step is to proceed with a displacement irrigation (the technic to be described later). By this means mucopus clumps are dislodged from cells and inaccessible spaces in the nose and brought out for ready inspection, in the collecting bottle. This irrigation, although it cannot localize the individual sinus or cell involved, gives valuable diagnostic confirmation. In many cases it does away with the necessity of sinus puncture in cases in which the patient is a child (sparing him the incidental trauma and fear).

In addition to the picture just described, many confirmatory details present themselves when a painstaking history and careful observations are the routine. Such suggestive findings as mouth breathing, nasal voice, alar irritation and eczema are frequent. All are due to obstruction and discharge. Loss of appetitite and failure to gain weight are common complaints. A high percentage of children with sinusitis suffer with otitis media, and the sinuses should be carefully studied when aural trouble is present. The cervical lymph nodes are always enlarged to some degree in these children, although the enlargement need not go beyond the "just palpable" stage.

A discussion of the diagnosis of nasal sinus conditions would not be complete without some mention of the use of transillumination and roentgen rays. Both of these otherwise valuable diagnostic aids fall short when used for children.

Because of the thin bones involved, transillumination is not accurate enough. The light seems to come through too easily, even in cases in which there is plenty of evidence of sinus involvement. Little is gained by its use on children.

In the usual case the roentgenogram is a great help in confirming the clinical diagnosis. Unfortunately (or perhaps fortunately) the pathologic condition in the type of sinusitis under discussion has usually not reached the stage at which mucosal thickening or osseous changes can be interpreted on the basis of roentgenograms. In my experience, roentgenograms have added little to the clinical picture in these cases; hence I do not use them as a routine. Their use is limited to selected cases.

TREATMENT

A method of treatment which can assist the function of the cilia and help the epithelium to clean and unburden itself could well be called a physiologic treatment. The method of cleansing and irrigation of the

sinuses described by and named after Proetz [2] gives this highly desirable physiologic treatment.

The Proetz displacement irrigation or any of the minor modifications recommended by other workers furnishes the rhinologist with a therapeutic measure that solves a great many theoretic and practical problems. Properly performed, it cannot add any trauma or insult to tissues already in a poor state for self defense. This advantage is inherent in its technic and in the irrigating solution used.

Intranasal instrumentation is practically eliminated. It is only occasionally necessary to use ephedrine solution for local shrinkage to give readier access to the irrigating fluid. This may be needed when the sphenoethmoid chink is narrowed or obliterated by edema. In this situation the preliminary use of a pledget of cotton, wet with ephedrine solution and gently applied where it is needed, opens the space sufficiently for the irrigating solution.

I find that a 0.5 per cent solution of ephedrine sulfate in physiologic saline solution, as recommended by Proetz, gives excellent results. A 0.125 per cent solution of neo-synephrin in physiologic solution of sodium chloride is satisfactory.

Used this way, in physiologic solution of sodium chloride and warmed to body temperature, the irrigating fluid has no appreciable irritating effect. Proetz [3] and Lierle and Moore [4] definitely showed the irritative possibilities of the solutions commonly used in the nose. They concluded that ephedrine in physiologic solution of sodium chloride is isotonic, nonirritating and slightly stimulating.

When the irrigating solution is caused to enter the sinus cavities by the displacement technic it mixes with the accumulated secretions. The products of mucosal reaction are diluted, made more fluid and less tenacious. The sticky, stringy mucopus is loosened from the mucosa and floated out or washed out through the ostiums into the common nasal cavity. The dilution and removal of the infectious products permits the ciliary function to carry on more efficiently and thereby greatly aids the mucosa in its natural processes of self cleansing.

From the practical point of view, in regard to its application in the treatment of children with sinus infections, the displacement technic offers the advantage of being entirely painless. The naturally fearful child sees no array of instruments. It is really the exceptional child who will not cooperate after a preliminary explanation and description

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2. Proetz, A. W.: The Displacement Method of Sinus Diagnosis and Treatment, St. Louis, Annals Publishing Company, 1931.

3. Proetz, A. W.: Effects of Drugs on Living Nasal Cilia, Ann. Otol., Rhin. & Laryng. **43**:450 (June) 1934.

4. Lierle, D. M., and Moore, P. M.: Effect of Drugs on Ciliary Activity, Arch. Otolaryng. **19**:55 (Jan.) 1934.

of what the physician is going to do. Once the attempt is made, the rest is easy, for by its simplicity and painlessness the procedure sells itself to the little patient.

The equipment needed for this treatment consists of a table, a source of suction, an irrigating solution, rubber tubing and a nasal suction tip. As the operator rapidly gains facility in the technic, other ideas will occur to him in the way of accessories and arrangements.

Although it is usually recommended that a reclining chair be used for the treatment, I have had satisfactory service from a sturdy, flat examining table. This table happens to be covered full length with a comfortable, thick pad of sponge rubber.

The source of suction may be either of two kinds of apparatus: (1) small electric pumps, of which there are a variety on the market, or (2) a water faucet suction outfit (Gray's or Frawley's). The electric

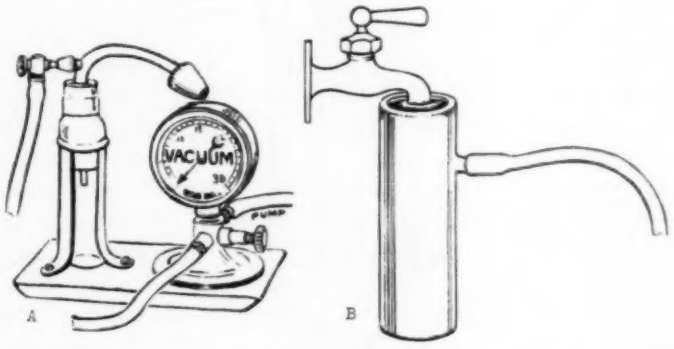

Fig. 1.—Suction apparatus for use in treatment of sinusitis by irrigation: *A*, vacuum gage for use with electric pump, and *B*, water faucet suction outfit.

pump (fig. 1 *A*) should have a gage attached, to permit adjustment of the suction to 180 mm. of mercury. Higher degrees of suction are not necessary and may give rise to discomfort. A water faucet suction outfit (fig. 1 *B*) is cheap and convenient. With this simpler apparatus a gage is not so necessary, for the usual office water supply is maintained at a fairly constant pressure which is about right for this purpose.

The simplest and most convenient nasal tip available is one that is found in every otolaryngologist's office. It is used with the handy suction bottle (fig. 2). The thumb hole readily allows the alternation of suction used in the treatment. When not in use the bottle stands in a special rest or in a simple block of wood, as illustrated.

Proetz has devised an ingenious combination of syringe for instilling the irrigating solutions and nasal tip for applying suction.

Before the irrigation is begun, the child is seated on the edge of the table and in a friendly, informal way is told about the treatment. He

is reassured as to its painlessness and rapidity. If the suction tip has never been used on him before, it is now demonstrated, first against his skin and then in his nostril.

The patient is placed on his back with his head hyperextended (off the table) so that the external auditory canal and the chin are in a vertical line. The back of the neck is carefully protected against the pressure of the table edge by any convenient form of pad. The table pad previously mentioned is thick enough and soft enough so that no additional protection is required. The mouth is kept fairly wide open, and the patient is instructed to breathe at a natural rate and rhythm through the mouth. He is also told to try hard not to swallow. It is often convenient to place an ordinary wooden tongue depressor, blade edgewise, across the mouth, between the back teeth. The patient bites

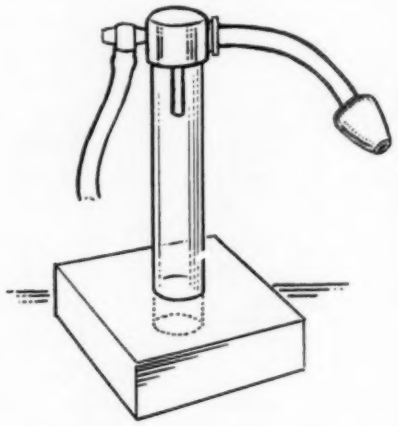

Fig. 2.—The suction bottle which is used with the nasal tip in irrigation of the sinuses. The bottle is shown standing in a drilled block of wood, as it does when not in use.

on this, and it helps to keep the mouth open, to minimize swallowing and to prevent the sudden ejection of the pharyngeal contents. Any form of simple mouth gag would probably be more efficient but would appear to the apprehensive child as a formidable instrument.

When the patient is in the proper position and has been told what to expect, 2 cc. of slightly warm solution is gently instilled into each nostril. It will cause less disturbance if allowed to flow down along the floor of the nose. Without loss of time, suction is applied at one nostril while the other is closed with a finger tip. After about a second, the suction is released by the removal of the thumb from the vent on the suction bottle. This cycle of suction for one second and release, or no suction, for two or three seconds, is repeated for eight to ten alternations.

The child is then allowed to sit up, relax and expectorate. Usually the procedure outlined is sufficient for a treatment, unless the amount of mucus found in the suction bottle seems to be excessive. If it is deemed necessary, the patient is again placed on his back and the entire procedure, which is simple and brief, is repeated.

It is best for the patient not to blow his nose for a few minutes after a treatment in order to give the irrigating solution a better chance to do its work. Treatments are given every second or third day at first, and the intervals are increased as progress indicates.

The suction applied at the nostril must be regulated to give no more than 180 mm. of negative pressure, and the apparatus should be one that permits this level to be reached quickly. The simple water jet suction fulfils these requirements beautifully.

Occasionally an adult patient mentions a transient headache after the treatment. This happens only after the first treatment or two and, according to my experience, almost never occurs in children.

It has been suggested on theoretic grounds that biologic preparations, such as phages or vaccines, can be instilled directly into the involved sinuses by this technic. I have not given this sufficient trial to warrant comparisons.

The instillation of antiseptics has not been shown to be of any value. In fact, from a physiologic point of view, such solutions are distinctly harmful to mucosal function.[5]

No discussion of the treatment of sinusitis in children can be complete without emphasis on the general care of the patient. The cooperation of the pediatrician is of the greatest importance. This must not be forgotten while the attention is focused on local therapy. A diet adequate in calories and vitamins, in cod liver oil, iron and other antianemic elements is invaluable. In short, anything that builds up the general health furthers eventual success.

CONTRAINDICATIONS

There does not seem to be any contraindication in children to this Proetz displacement treatment. I have not had a single untoward outcome or complication ascribable to the procedure or solution used.

On theoretic grounds the fear has been expressed that the middle ear is vulnerable to approach via the eustachian tube. This has never happened in my cases. Fox and his co-workers,[6] using practically the same technic, had only 2 unimportant aural infections result. These were nonsuppurative and ascribed to swallowing during the treatment. There were no further occurrences in later cases after patients were warned not to swallow.

5. (*a*) Proetz.[3] (*b*) Lierle and Moore.[4]

6. Fox, N.; Harned, J. W., and Peluse, S.: Irrigation by Posterior Suction. Arch. Otolaryng. **29**:263 (Feb.) 1939.

Proetz,[2] in his monograph on the displacement method, said:

> The eustachian tube . . . does not enter into the question of displacement. Owing to the elastic nature of its pharyngeal end, to its length and to the relatively small air content of the middle ear, no air is displaced from it and no fluid enters it, no matter in what position the head may be placed. . . .
>
> . . . no solution has ever been found in radiograms to have penetrated the middle ears or even the tubes.

REPORT OF CASES

The following case records are illustrative of the conditions already discussed, of their treatment as described and of the results obtained.

CASE 1.—E. F., a school girl aged 10 years, was referred to the otolaryngologic clinic from the pediatric department on Feb. 7, 1939, with a history of frequent head colds, especially during the last two years, nasal discharge, cough and recent acute purulent otitis media.

Findings.—There was a profuse mucopurulent nasal discharge anteriorly and posteriorly, as well as secondary granular pharyngitis with lateral bands.

Treatment and Course.—A series of Proetz irrigations of the sinuses was started. Treatment was given on February 9 and 14, and much less nasal discharge was noted. At the time of treatment on February 21, March 14 and March 28, slight increase in "phlegm" for a few days was reported, but there were no other complaints. Further irrigations were done on April 4 and 11; the nose was clean, and there were no complaints.

On June 13, 1939, a follow-up examination showed the patient's condition to be satisfactory, and there were no complaints. Three irrigations were given after April 11, because her mother wanted the patient to have them. She had only one slight cold during the course of irrigations, and that responded promptly. The mother states that all previous colds lasted for weeks and were followed by protracted cough and bronchial trouble.

CASE 2.—F. R., a boy aged 11½ years, was referred to the otolaryngologic clinic from the pediatric department on Dec. 8, 1937, with a history of frequent colds, nasal obstruction and discharge. The boy had gone through several rather severe infections of the respiratory tract, having once been reported as suffering from "patch in the upper lobe of the right lung." (He was not then under the care of the New York University clinic.) This anomaly was felt to be beginning bronchiectasis secondary to sinusitis on an allergic basis. Because of cough and repeated respiratory episodes, roentgenograms of the chest were taken in November 1937. No infiltration or consolidation was found. Calcific nodes were seen. The reaction to the Mantoux test was normal. At another time a moderately positive reaction to a cutaneous test for allergy to house dust was reported.

All through 1937 the patient had complained of headaches and nasal stuffiness and had been treated routinely with ephedrine and neo-silvol drops.

Findings.—On Dec. 8, 1937, examination on admission to the clinic showed engorged turbinates, red mucosa, slight crusting of nasal discharges and postnasal drip.

Treatment and Course.—A series of Proetz irrigations of the sinuses was started. Treatment was given on December 9 and 14, and the report was given that the nose was much clearer than it had been for a long time. After three more treatments, Dec. 16 and 28, 1937, and Jan. 6, 1938, it was noted that there had been no headaches and no coughing. At the time of the irrigation on January 20,

there was a cough which had been present for the last three days. On January 27, the date of the next treatment, it was reported that relief was prompt after the preceding one. Another irrigation was given on February 10, and there were no complaints. Again at the time of treatment on February 24 and March 10 there were no complaints; results of an examination were essentially negative, and the patient was discharged.

The pediatric check-up note of May 19, 1938, stated that the postnasal drip was present, but Dr. Berlinrood added that it was slight and needed no more treatment than the occasional use of ephedrine drops at home. Dr. Berlinrood also reported that there was no longer any complaint of, or need of treatment for, the severe protracted colds which had originally brought the boy to the pediatric department. The boy is doing well generally.

CASE 3.—E. L., a school boy aged 11 years, was referred on Dec. 27, 1938 because of a disturbing cough and postnasal drip of about one month's duration. There was a history of many similar previous attacks over a number of years.

Findings.—There was a moderately profuse mucopurulent discharge throughout the nose. Congested turbinate bones and a granular lymphoid pharynx with lateral bands were observed. The chest was clear.

Treatment and Course.—A series of displacement irrigations was started, the treatments being given at about two or three day intervals. On Jan. 4, 1939, the date of his third visit, the boy stated that he coughed much less. On January 27, the time of his fifth visit (sixteen days after the preceding one), the nasal condition showed marked improvement in that the discharge was greatly diminished and the mucosa clean.

CASE 4.—D. F., a boy aged 5½ years, was referred on Jan. 2, 1939 with a history of continuous nasal discharge for the past two winters, as well as cough and postnasal drip. The boy was progressing poorly, with little appetite and little or no gain in weight. The tonsils and adenoids had been removed when he was 5 years of age and the adenoids again in the summer of 1938. The mother stated that the sinuses were "cloudy" in a roentgenogram taken in August 1938. The chief reason for the parents' and the physician's anxiety was the patient's failure to respond to the usual antibechic measures.

Findings.—The nose was full of mucopurulent material. The turbinate bones filled the anterior part of the nose. The pharynx was granular and showed lateral bands. The chest was clear.

Treatment and Course.—A series of displacement irrigations was started. On Jan. 18, 1939 the mother reported "no more cough" and on February 17 "doing fine. Others have noticed improvement in his appearance." The last treatment (in a total of nine) was given on Feb. 27, 1939, at which time the nose was quite clean and all the original complaints had been cleared up.

On March 22, 1939 there was a mild recurrence due to a fresh cold. This responded promptly to two irrigations.

CASE 5.—P. B., a boy aged 8½ years, was referred on Oct. 12, 1938 with a history of "recent severe headaches and crankiness, probably due to sinusitis." Seasonally he always suffered from frequent colds which were characterized by profuse nasal discharge and cough rather than the headaches of the current episode. Attacks were usually associated with cervical lymphadenitis and a low grade fever (a temperature of from 99 to 100 F.). "Involvement of all the sinuses, especially the ethmoid," was the substance of a verbal roentgenographic report, but the roentgenograms were not available.

Findings.—The nasal mucous membrane was engorged, and there was profuse mucopurulent discharge anteriorly and posteriorly. No other obvious cause for headache was observed. The chest was clear.

Treatment and Course.—A series of displacement irrigations was started, and treatments were given at about two or three day intervals. On Oct. 21, 1939 the patient reported "no headaches at all." They had diminished greatly in severity and persistency after the second treatment. The nasal discharge was much diminished. On December 9, the child had only a slight "running nose," which cleared up by the time of the next visit. On Dec. 12, 1938 the eleventh and last treatment was given. Later reports showed no more of the usual heavy colds that winter. It was reported that the occasional slight cold responded quickly to the nasal instillation of a 1 per cent solution of ephedrine sulfate in physiologic saline solution, administered with the patient's head in the Proetz position.

Case 6.—E. B., a boy aged 7 years, was referred on Dec. 2, 1938 with a history of frequent colds associated with nasal discharge and cough and of postnasal discharge practically "always." The present condition was of about one month's duration, and the mother was worried because of the child's increasing anorexia. The cough did not respond to the usual remedies or to steam inhalations.

Findings.—There was a moderate amount of mucopurulent postnasal discharge together with the typical reaction in the pharynx in the form of lymphoid islands and lateral bands. The chest was clear.

Treatment and Course.—Six sinus irrigations by displacement were given up to Dec. 23, 1938, at intervals of three or four days. On December 23 the mother reported that the cough had ceased completely, having subsided rapidly since the third treatment. There was still a slight postnasal drip, which diminished to the vanishing point by the next and last visit, on Jan. 4, 1939.

Case 7.—J. A., a boy aged 10 years, was referred on Sept. 17, 1938 because of a severe cough of more than a month's duration. All the usual antibechic measures had been fruitless. The child was pale and beginning to feel weak from the long siege. His appetite was poor, and there was morning nausea. There was a history of frequent and protracted colds over a period of several years. The mother said she thought that the boy's temperature was often moderately elevated.

Findings.—The nasal tissues were swollen and water-logged. There was a moderately profuse, thin, mucopurulent discharge, which showed no evidence of eosinophilia when smears were made and examined under the microscope. The pharynx presented well marked lateral bands.

Treatment and Course.—Three treatments during the first week, with the usual measures of shrinking and tamponade, had no effect whatever in diminishing the complaints. From Sept. 28 to Oct. 7, 1938 the patient received five displacement irrigations of the sinus, which were followed by prompt easing of the cough. On October 7 the mother reported, "The cough is much better and the boy is improving in every way." On October 19, after two more irrigations, there was no cough and practically no postnasal discharge. The child was gaining rapidly in weight and his general condition was improving.

On Jan. 16, 1939 the mother brought the boy in for treatment of a bad cough. He had been entirely free of winter complaints since his last visit. Although the mother hoped for a repetition of the earlier success, the history and the absence of local findings suggested pertussis, which the condition proved to be. Irrigation treatment, of course, was not indicated or used.

CASE 8.—R. D., a boy aged 11 years, was referred to the otorhinolaryngologic clinic from the pediatric department on April 9, 1936 because of an acute exacerbation of chronic otitis media and a history of frequent colds and attacks of grip associated with postnasal discharge and cough. The aural condition healed by April 28, 1936, the nasal condition remaining in chronic status quo.

The patient was again referred to the clinic on Oct. 12, 1937.

Findings.—The nose was found to be full of mucopus, anteriorly and posteriorly.

Treatment and Course.—A series of Proetz irrigations was started. Treatments were given on Oct. 12, 19 and 21, 1936. Another irrigation was done on October 26; later the parent reported that twenty-four hours after that treatment, while in school, the boy had a headache, vomited and then lost his appetite for two days. There had been no complaints after previous treatments and none during the afternoon and evening immediately following the treatment in question. The sinus irrigation was probably not the cause of these complaints after a twenty-four hour interval; his parent said, "He always has been fussy about food and vomits readily." On November 4 further treatment was given, and no such reaction was reported as that occurring after the treatment of October 26. The child was showing definite improvement. The treatments were given on November 9, 11, 18 and 30. On December 7 return of the purulent discharge was noted, in connection with an acute cold. On December 9 the condition was again satisfactory. On December 16 there were again complaints of anorexia and upset stomach "because of treatment."

Report on the pediatric examination on Dec. 16, 1937 follows: "The child cannot localize discomfort or complaint. He is of a nervous disposition, often has nightmares and is jumpy by day. The lungs are clear and the heart normal. The cervical glands are small. There is no anemia, although the child is pale."

Later pediatric notes refer to occasional slight nasal complaints. There have been repeated reports of "developing very well," although "failure to gain" has been a regular complaint. The boy became too old for the pediatric clinic, and contact was lost.

CASE 9.—H. B., a girl aged 3½ years, was referred on April 28, 1936 because of a long-standing nasal discharge and obstruction. Nights were uncomfortable owing to these complaints. There was also considerable anorexia.

Findings.—The nasal mucosa was congested and smeared with mucus. The tonsils had been adequately removed one year ago. Before that, there were frequent bad colds, to which the parents ascribed the present condition.

Treatment and Course.—The first attempt at Proetz irrigation was unsuccessful; the child would not cooperate. (This was one of the first patients on whom this treatment was used.) At the next visit, May 11, 1936, the irrigation was entirely successful. Another treatment was given on May 18, 1936. On May 25, 1936 good improvement was noted. On June 8, 1936, after a total of five irrigations, the patient was discharged symptom free.

CASE 10.—P. G., a girl aged 7 years, was referred on Oct. 31, 1938 because of a severe cold that had lasted more than a month. This was associated with a postnasal discharge and an incessant cough, especially at night. The cough made rest at night impossible for the patient and her family. The usual cough prescriptions and steam inhalations produced no results. There was a history of frequent colds for the past three or four years.

Findings.—The nasal cavities contained a moderate amount of rather thin mucopurulent discharge, especially posteriorly. The nasal mucosa was moderately engorged. The pharynx presented the typical lateral bands. The chest was clear.

Treatment and Course.—By the third sinus displacement irrigation, on Nov. 9, 1938, the mother reported that the cough was almost gone. After a total of five irrigations the child was entirely symptom free, and the nose was clean.

CASE 11.—W. D., a boy aged 6 years, was referred on Dec. 1, 1937. He had a congenital cardiac lesion associated with tremendous enlargement. The complaints on admission were: (1) frequent colds with cough, (2) anorexia, (3) postnasal discharge and (4) signs suggestive of allergy.

There was a history of aural trouble in September 1937. The child had previously had several courses of nasal tampon treatment by other physicians.

Findings.—The nose was full of purulent crusts, and there was evidence of recent bleeding in the left nostril. The turbinates were engorged. There was mucopurulent postnasal discharge together with secondary granular pharyngitis. A smear of the discharge showed all the cells to be polymorphonuclears; there were no eosinophils.

Treatment and Course.—A series of Proetz sinus irrigations was started. On December 6 there was much less discharge in the nose. On December 15 the mother reported that after the last two treatments the child felt tired and sleepy. Dr. Bien, the pediatrician who had referred the child to me, checked the heart before and after the current treatment and could find no ill effects. The parent was advised, in view of the child's congenitally defective heart, to be stricter in curtailing his activities, which were strenuous even for a normal boy, and it was found that the child did not complain again. On Dec. 27, 1937, after seven irrigations, the child came in with a fresh acute cold. The usual irrigation was given, and by the next visit, on Jan. 3, 1938, the cold was gone and excellent progress reestablished. The purulent condition in the nose had entirely cleared up by this time. The discharge was now a clear watery mucus. A smear showed a great many eosinophils, indicating the allergic basis for the entire condition (obstruction to ventilation and drainage leading to secondary infection). A check-up from the allergic angle was advised. This apparently was not actively carried out.

Just a year later, on Dec. 27, 1938, the boy was brought in again, after an infection of the upper respiratory tract. Examination revealed the same findings as before but to a much more moderate degree. Three irrigations in eight days controlled the symptoms, and then a few treatments at weekly intervals brought about a satisfactory condition in the nasosinal tract, complete relief of symptoms being obtained.

COMMENT ON CASE REPORTS

A first reading of the case reports would suggest that the duration of treatment is not so much shorter than the time necessary in other methods. A closer examination will point out the gratifying promptness with which long-standing complaints respond to these irrigations. This refers of course chiefly to the subjective complaints. Profuse nasal discharges, clogged nasal airways and distressing coughs of long standing are relieved by a few treatments. The resulting comfort and restful nights are highly appreciated.

The confidence gained by the success so far affords an opportunity to attack the problem of the underlying pathologic condition with less urgency and worry. The necessary general hygienic measures become much more effective when the local burden is lightened by adequate, sustained ventilation and drainage.

Recurrences tend to diminish in frequency, and the relative mildness of the new attacks is notable. Usually few treatments are then necessary.

The more objective evidence of sinus trouble does not disappear so readily. This is to be expected, and the treatments must be continued.

PROGNOSIS

The displacement irrigation treatment for sinusitis has given satisfactory clinical results in cases in which the condition is properly diagnosed. Complaints of long duration can be relieved in a relatively short period of treatment. It is felt that each patient, in a sense, is his own control, in that almost every case history tells of long extended previous attacks. The occasional recurrences in the treated patients respond promptly to an irrigation or two. This contrasts with the former experience of renewed prolonged distress with each reinfection.

No claim of permanent cure can be even suggested without more study and follow-up. On the other hand, the prognosis is favorable for a clinical cure based on gratifying subjective relief and local findings.

SUMMARY

1. Sinusitis in children is discussed from the viewpoint of diagnosis, of conservative physiologic treatment and of results obtained.

2. Emphasis is placed on the necessity for the more frequent recognition of sinusitis in children.

3. The treatment described (irrigation of the sinuses by displacement) is simple and painless; it does not further the popular dread of disease and treatments of the sinuses and is in accord with all present concepts of the function of the nasosinal epithelium. The same procedure can also be of diagnostic value.

4. The treatment as described gives early, satisfactory relief to children suffering from nasal sinus infections. The secondary cough responds particularly well to the removal of its cause. The treatment greatly reduces the duration and frequency of disability of the upper respiratory tract.

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INTRACRANIAL PATHWAYS OF INFECTION FROM DISEASES OF THE SPHENOID AND ETHMOID SINUSES

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The pathologic process and the pathways of intracranial infection from the paranasal sinuses have not been investigated as routinely or as thoroughly as those of the mastoid bone and petrous pyramid. Except for gross examination at the necropsy table, little effort has heretofore been made to study the sinuses microscopically even in cases of bacterial meningitis of undetermined pathogenesis. Since in only a small percentage of instances is the lesion visible macroscopically, a large number of lesions starting in the sinuses have undoubtedly been overlooked and the primary foci not discovered. It was once assumed that in the presence of suppuration of the middle ear the meningitis was otitic in origin even though pathologic proof of this origin did not exist.

During the past seven years, we have been removing the paranasal sinuses en bloc in all cranial autopsies in cases of meningitis. The block consists (fig. 1) of the sphenoid sinus and basisphenoid bone, the ethmoid labyrinth and the region of the cribriform plate. The superior and middle turbinates are included. The frontal sinuses and antrums are routinely opened and investigated, but are not included in this study, since serial section of so large a block is not feasible. The block is neither opened nor probed, but after gross inspection it is decalcified and then studied in serial sections which are cut coronally (fig. 2). On only three occasions did we find gross evidences of bone necrosis and an obvious source of infection.

We have been able to demonstrate microscopically the origin of the infection in the sphenoid and ethmoid sinuses and to trace the spread to the meninges in many cases of so-called cryptogenic bacterial meningitis, or that of undetermined origin. In other instances, the assumption that the meningitis was due either to a coexisting mastoiditis or to pneumonia was proved to be wrong and the true focus of infection in the sinuses established.

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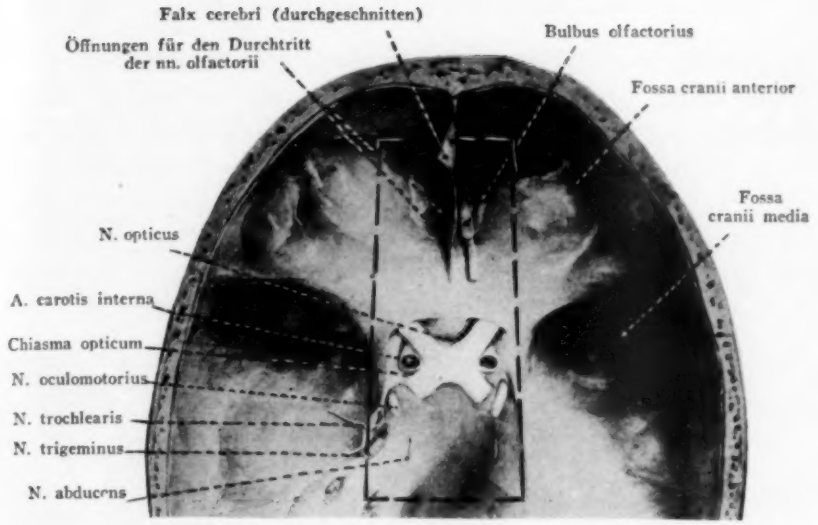

Fig. 1.—Dorsal view of the base of the skull, showing the line of incision for removal of ethmoid and sphenoid sinuses en bloc. The nasopharynx is entered through the clivus with a chisel. The petrous tip and the lesser and the greater wing of the sphenoid bone are severed. The frontal bone is then cut or sawed through and the periosteum of the orbit separated from the lateral wall of the ethmoidal labyrinth. The crista galli is split anterior to the cribriform plate. (Spalteholz, W.: Hand Atlas der Anatomie des Menschen, ed. 7, S. Hirzel, Leipzig, 1913).

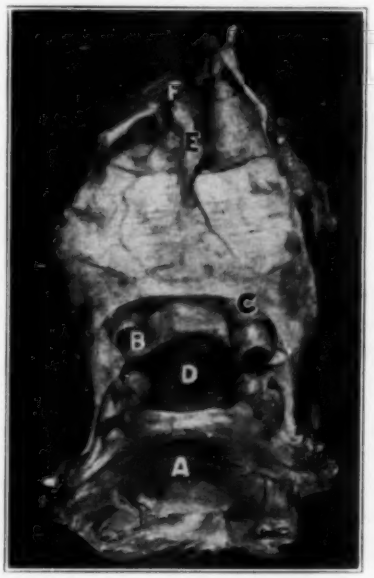

Fig. 2.—Appearance of the sinus block after removal: *A*, clivus; *B*, internal carotid artery; *C*, optic nerve; *D*, sella turcica; *E*, crista galli, and *F*, cribriform plate.

DETERMINATION OF PATHWAYS OF INFECTION

The finding of polypi or pus in the sphenoid or ethmoid sinuses cannot be regarded as proof that the meningitis originated in these foci. Several potential foci of suppuration may be present simultaneously, viz., such conditions as otitic infection, sinusitis and pneumonia. Unless there is a gross perforation with bone necrosis and a noticeable dehiscence, microscopic evidences of spread must be demonstrated. The location of the meningeal exudate at the base of the skull is no indication as to the

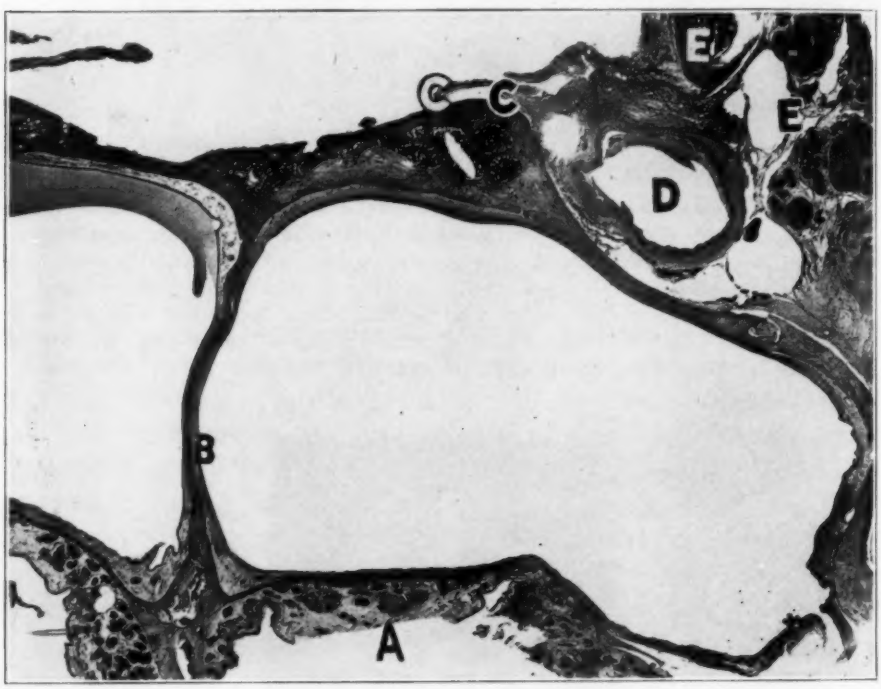

Fig. 3.—Section through a normal sphenoid sinus, showing: *A*, anterior wall of sphenoid and nasopharyngeal mucosa; *B*, mucosa lining the intersphenoid septum; *C*, posterior wall with attached dura; *D*, internal carotid artery, and *E*, cavernous sinus with its nerve content.

origin of the infection except in a rare case in which the meningitis is localized to the olfactory bulbs and the surrounding tissues.

Through study of serial sections the infection can be traced from its origin in the mucosa of the sinuses to the cranial contents. Some of the pathways of infection from the sinuses to the cranial cavity have been demonstrated: (1) direct extension through the bony wall of a diseased

sinus by osteomyelitis (Turner and Reynolds,[1] Hesse,[2] Kramer and Som[3]), (2) lymphatic extension via perineural lymph sheaths, (Jacobsgaard;[4] Turner and Reynolds[5]) and (3) venous spread by perforating vessels to the cavernous sinus (Hajek[6]). In addition, the following means of spread have been mentioned without proof, (4) congenital dehiscences and (5) metastasis during a sepsis.

In our own series we were able to demonstrate microscopic evidences of spread to the meninges by the following pathways: (1) osteomyelitis and osteitis, (2) lymphatic extension via perineural lymph sheaths, (3) lymphatic extension via perivascular lymph channels, (4) vascular spread through perforating vessels to the cavernous sinus, (5) direct invasion of the meninges through congenital dehiscences and (6) spread through a persistent craniopharyngeal pouch.

ANATOMY

A brief discussion of the anatomy of the sphenoid and ethmoid sinuses is essential to the proper understanding of the pathogenesis.

The mucosa of the sphenoid and ethmoid sinuses is separated from the cranial cavity by a common bony wall (fig. 3). This bony partition is of varying thickness and structure. The roof of the ethmoid sinus is composed almost exclusively of compact lamellar bone. Similarly the roof and walls of the sphenoid sinus may be formed by lamellar bone when the sinus is well pneumatized. Just as frequently, however, cancellous

1. Turner, A. L., and Reynolds, F. E.: Study of Paths of Infection to Brain, Meninges, and Venous Blood Sinuses from Neighboring Peripheral Foci of Inflammation: Acute Suppuration in the Accessory Sinuses; Cavernous Sinus Thrombosis; Acute Leptomeningitis; Death; Autopsy, with Microscopic Examination of Ethmoidal and Sphenoidal Air Sinuses and Cavernous Blood Sinuses, J. Laryng. & Otol. **43**:565 (Aug.) 1928.

2. Hesse, W.: Zur Ätiologie der Keilbein-Osteomyelitis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:94, 1933.

3. Kramer, R., and Som, M. L.: Actinomycosis of the Sphenoid with Actinomycotic Meningitis and Brain Abscess, Ann. Otol., Rhin. & Laryng. **44**:973 (Dec.) 1935.

4. Jacobsgaard, J.: Pathologisch-anatomische Beiträge zur Frage nach den Fortleitungswegen der rhinogenen Meningitis, Ztschr. f. Laryng., Rhin., Otol. **26**: 387, 1936.

5. Turner, A. L., and Reynolds, F. E.: Study of Paths of Infection to Brain, Meninges, and Venous Blood Sinuses from Neighboring Peripheral Foci of Inflammation: Nasal Mucous Polypi; Intranasal Operation on the Ethmoid Sinuses; Purulent Leptomeningitis; Death; Autopsy, with Microscopic Examination of Olfactory Nasal Mucous Membrane, Ethmoidal and Sphenoidal Air Sinuses and Meninges, J. Laryng. & Otol. **42**:525 (Aug.) 1927.

6. Hajek, M.: Ein Beitrag zum Studium des Infektionsweges bei der rhinogenen Gehirnkomplikation, Arch. f. Laryng. u. Rhin. **18**:290, 1906.

or spongy bone separates the sphenoid sinus from the sella turcica. The floor of the sphenoid sinus is formed by cancellous bone containing red or yellow marrow.

In compact bone, venules from the mucoperiosteum of the sphenoid sinus pierce the bone and communicate with one of the larger cranial sinuses or with dural vessels. These perforating venules are surrounded by perivascular spaces which are probably lymph channels communicating with the cranial contents.

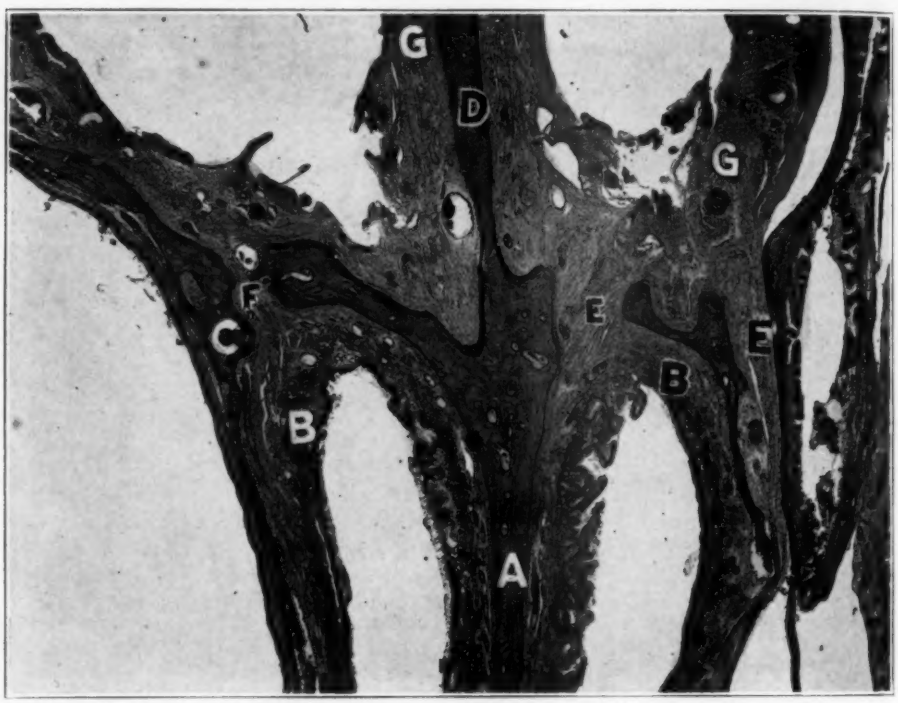

Fig. 4.—Section through a region of a normal cribriform plate, showing: *A*, nasal septum; *B*, olfactory mucosa; *C*, attachment of the middle turbinate bone; *D*, crista galli; *E*, olfactory fibers passing through the cribriform plate; *F*, perforation of the cribriform plate, and *G*, dura.

In cancellous bone, the deeper layer of the mucoperiosteum sends at intervals microscopic digital extensions of connective tissue, with or without vessels, into the substance of the bone. These projections reach into the marrow spaces. From the marrow spaces diploic veins communicate either with tributaries or directly with the cavernous sinus.

The aforementioned perivascular spaces and periosteal extensions furnish avenues for spread of infection from the mucosa to the bone and cranial cavity.

There are some twenty filaments of the olfactory nerve situated in the upper third of the nose in the nasal mucosa, covering the septum and the superior and middle turbinates (fig. 4).

Zwillinger,[7] among others, injected dye into the subarachnoid spaces and filled the perineural lymph spaces in the olfactory mucosa. The dye was further suffused into the superficial lymphatic network of the olfactory mucosa. This established the continuity between the subarachnoid space, the perineural sheaths and the lymphatic network of the olfactory mucosa.

Fig. 5.—Gross appearance of the posterior surface of the sphenoid sinus and clivus, showing macroscopic perforation. There was a free communication between the cavity of the sphenoid sinus and the subarachnoid space.

PATHWAYS OF INFECTION

Osteitis and Osteomyelitis.—In the course of acute inflammation of the sphenoid and ethmoid sinuses, osteitis of the lamellar bone forming both the os planum and the roof of the ethmoid and the sphenoid sinus may result. This compact bone, being thin and lacking red marrow, may undergo local necrosis by contiguity. Microscopically, such bone shows loss of lamination, disappearance of bone corpuscles and peripheral

7. Zwillinger, H.: Die Lymphbahnen des oberen Nasenabschnittes und deren Beziehungen zu den perimeningealen Lymphräumen, Arch. f. Laryng. u. Rhin. **26**: 66, 1912.

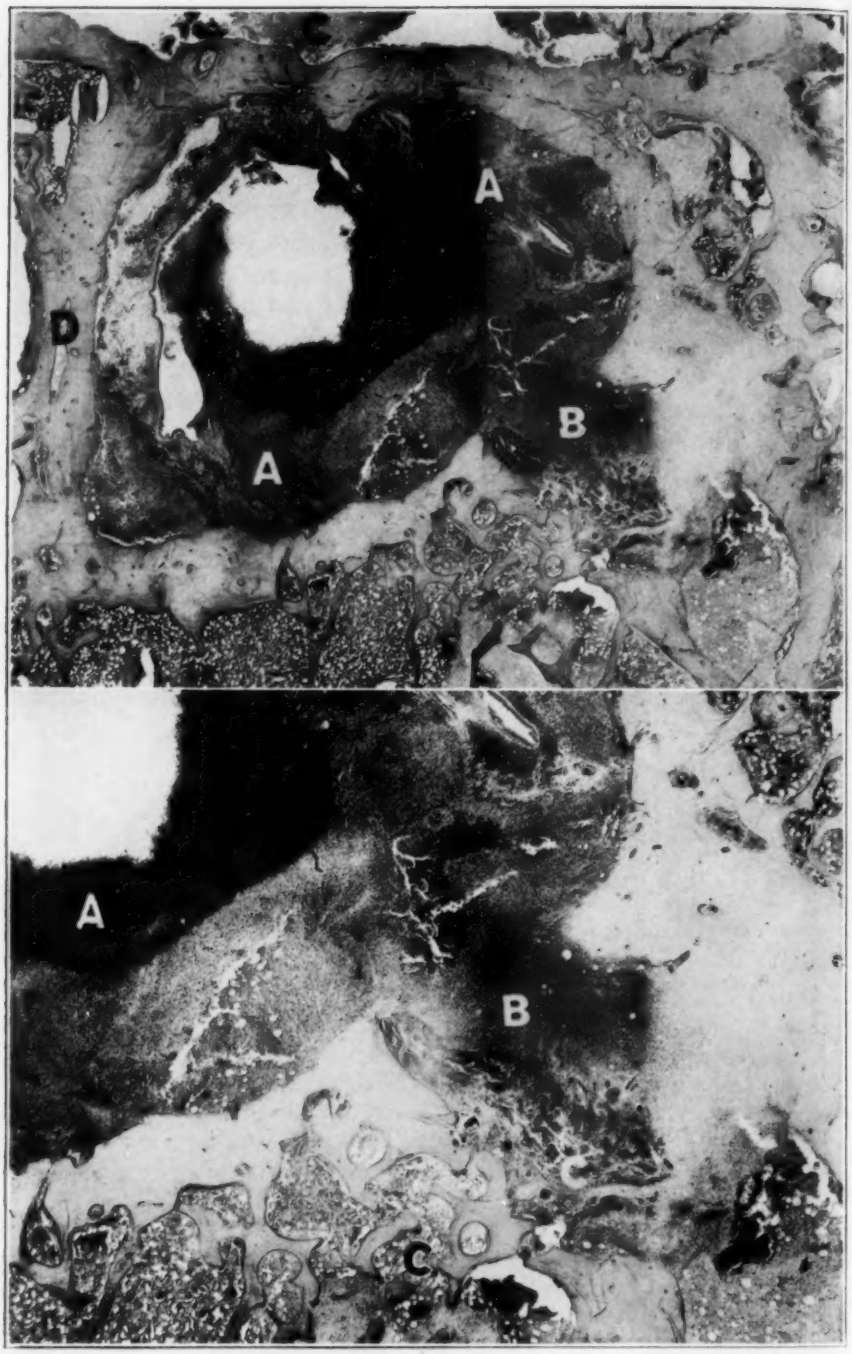

Fig. 6.—Section through a sphenoid sinus. The upper portion shows severe inflammation of the mucosa and invasion of the bone marrow: *A*, inflamed mucosa; *B*, spread along a preexisting mucoperiosteal extension; *C*, inflamed dura, and *D*, intersphenoid septum. The lower portion, made under higher power, shows an area of osteomyelitis: *A*, inflamed mucosa; *B*, zone of invasion, and *C*, osteomyelitis.

erosion. Progressive erosion may eventually result in actual gross dehiscence and in the establishment of a direct connection between the sinus and the dura.

We have observed 3 such cases of gross perforation of the sphenoid or ethmoid sinuses. These are the only instances in which macroscopic examination alone was sufficient to reveal the portal of entry. Figure 5 illustrates an inflammatory defect in the posterior wall of the sphenoid sinus, together with dural perforation. Direct communication between the subarachnoid space and the sphenoid sinus was present. The patient had cerebrospinal rhinorrhea. The increased intracranial tension may have produced a blow-out of the dura, which already was weakened by the underlying osteomyelitis and sequestration of bone (fig. 11, lower part).

When infection involves cancellous bone, osteomyelitis results. After initial infection of the marrow spaces, secondary inflammation of the meninges may be brought about in one of two ways: (1) Thrombophlebitis of a perforating vein may carry the infection to the dura, or (2) progressive bone necrosis may result in epidural suppuration. The establishment of this initial infection of the marrow spaces has been seen to occur in one of three ways: (1) direct spread into the bone from the inflamed mucosa along the preexisting mucoperiosteal extensions (fig. 6), (2) formation of a subperiosteal abscess with direct exposure of the bone to suppuration (fig. 8) or (3) extension of a microscopic purulent phlebitis from the mucosa into the marrow spaces (fig. 9).

In most of our cases of osteomyelitis we have found a chronic submucosal abscess at the primary site of infection. Extension to the bone occurred after an acute exacerbation of the chronic inflammation. Such submucosal abscesses are not uncommon in the sphenoid and ethmoid sinuses and should be regarded as a possible menace to the patient. Microscopically, a distinct fibrotic capsule usually encloses the area of suppuration, which is densely packed with polymorphonuclear leukocytes, lymphocytes and plasma cells. At one area in the capsule, there may appear a break in the fibrous tissue, due to acute inflammation. From this site invasion of the bone takes place.

The chronicity of the abscesses may be important in helping to establish the pathogenesis of the infection in cases of cavernous sinus thrombosis. Serial sections in an instance of cavernous sinus thrombosis resulting from a furuncle of the nose associated with phlebitis of the angular and ophthalmic veins showed severe osteomyelitis of the basisphenoid bone as well as numerous acute abscesses in the mucosa of the sphenoid sinus. These abscesses were the result of retrograde

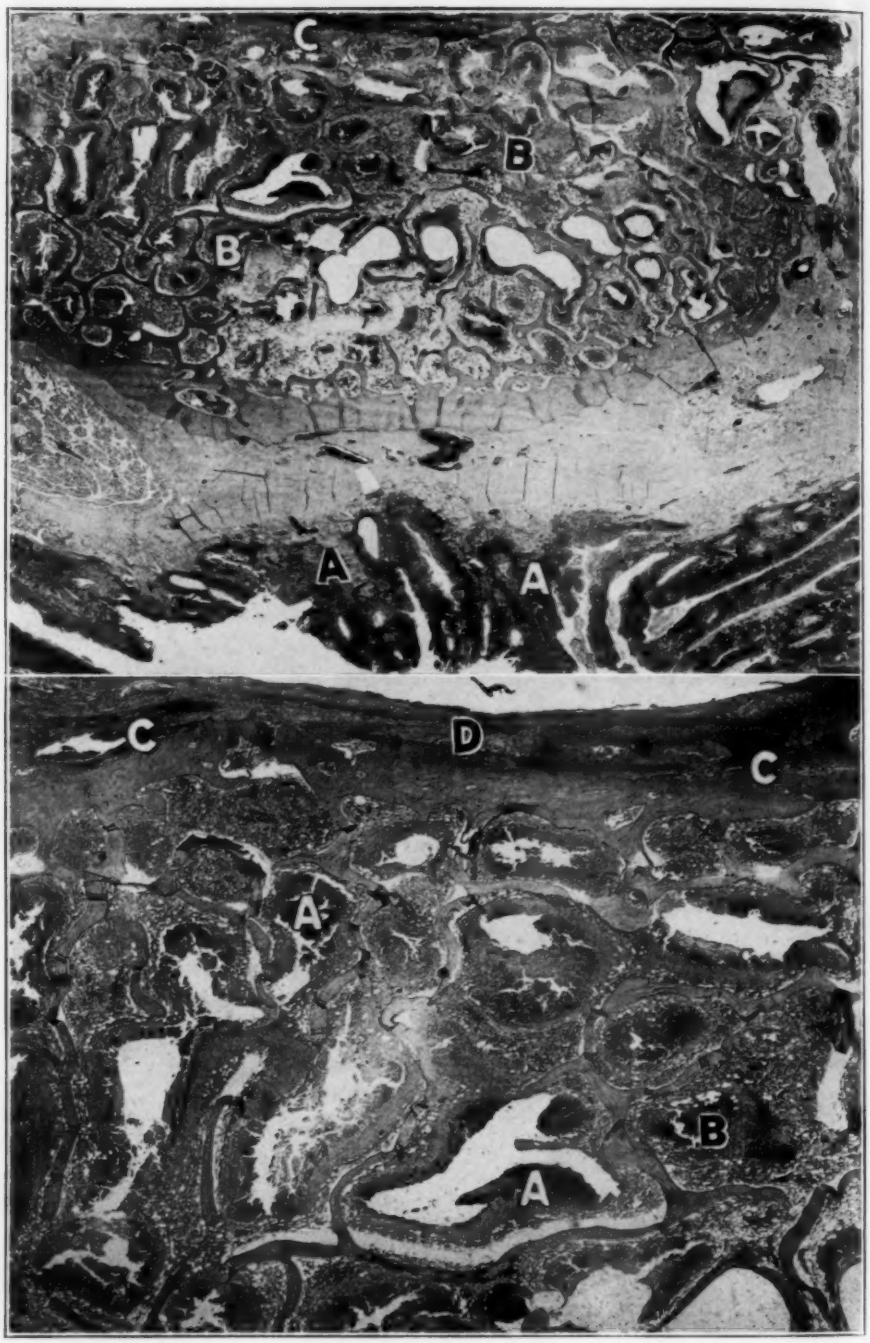

Fig. 7.—A section of the basisphenoid bone. The upper portion shows: *A*, nasopharyngeal mucosa; *B*, osteomyelitis, and *C*, adherent dura. The lower part, made under higher power, shows: *A*, osteomyelitis; B, focal abscess in the bone marrow; *C*, epidural abscess, and *D*, inflamed dura.

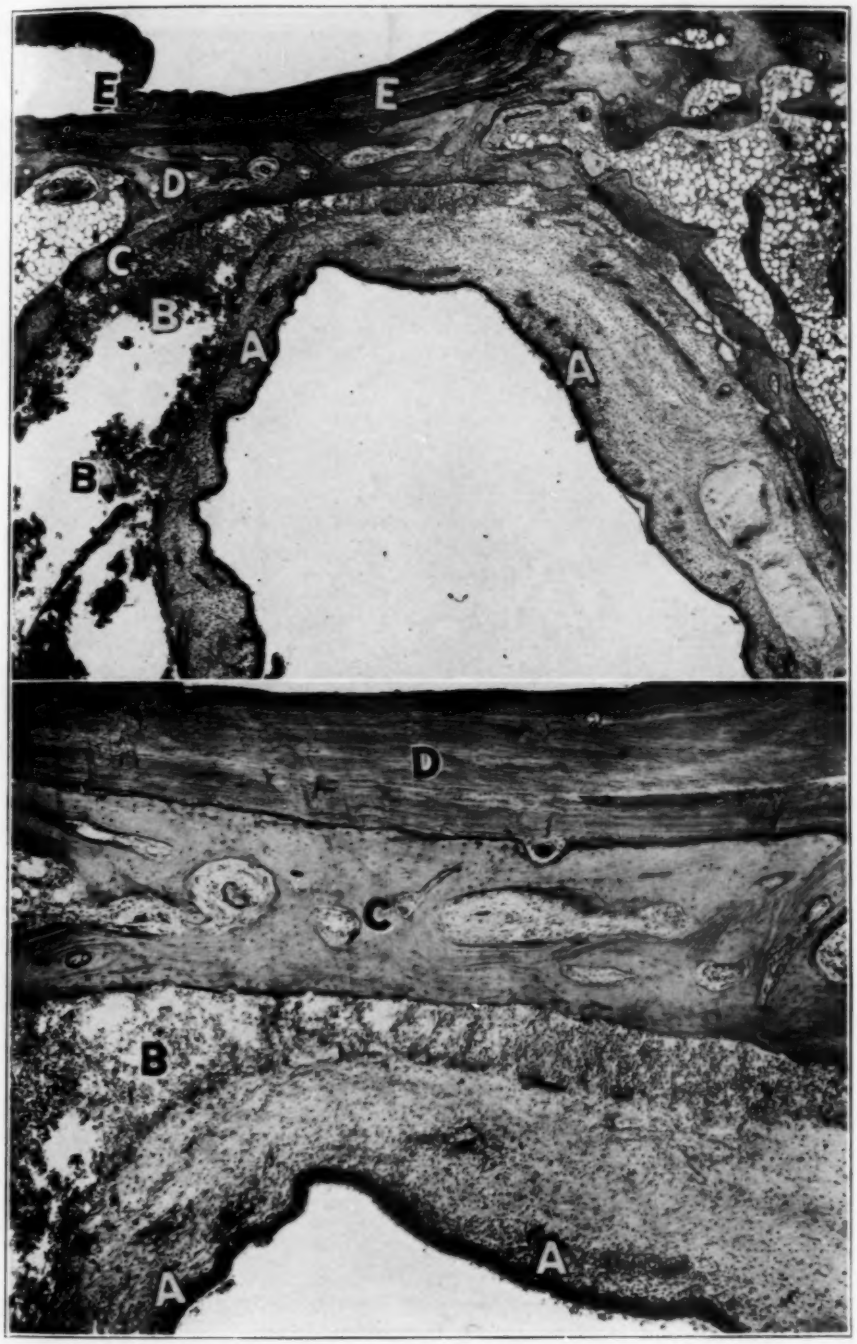

Fig. 8.—A section through the posterior wall of the sphenoid sinus. The upper portion shows a subperiosteal abscess with direct exposure of the bone to suppuration: *A*, mucosa; *B*, subperiosteal abscess; *C*, site of invasion of bone; *D*, osteomyelitis in the posterior wall of the sphenoid sinus, and *E*, inflamed dura. The lower portion, made under higher power, shows a subperiosteal abscess causing separation of the mucosa from the bony wall. *A*, mucosa; *B*, subperiosteal abscess; *C*, posterior wall of the sphenoid sinus, and *D*, markedly thickened dura.

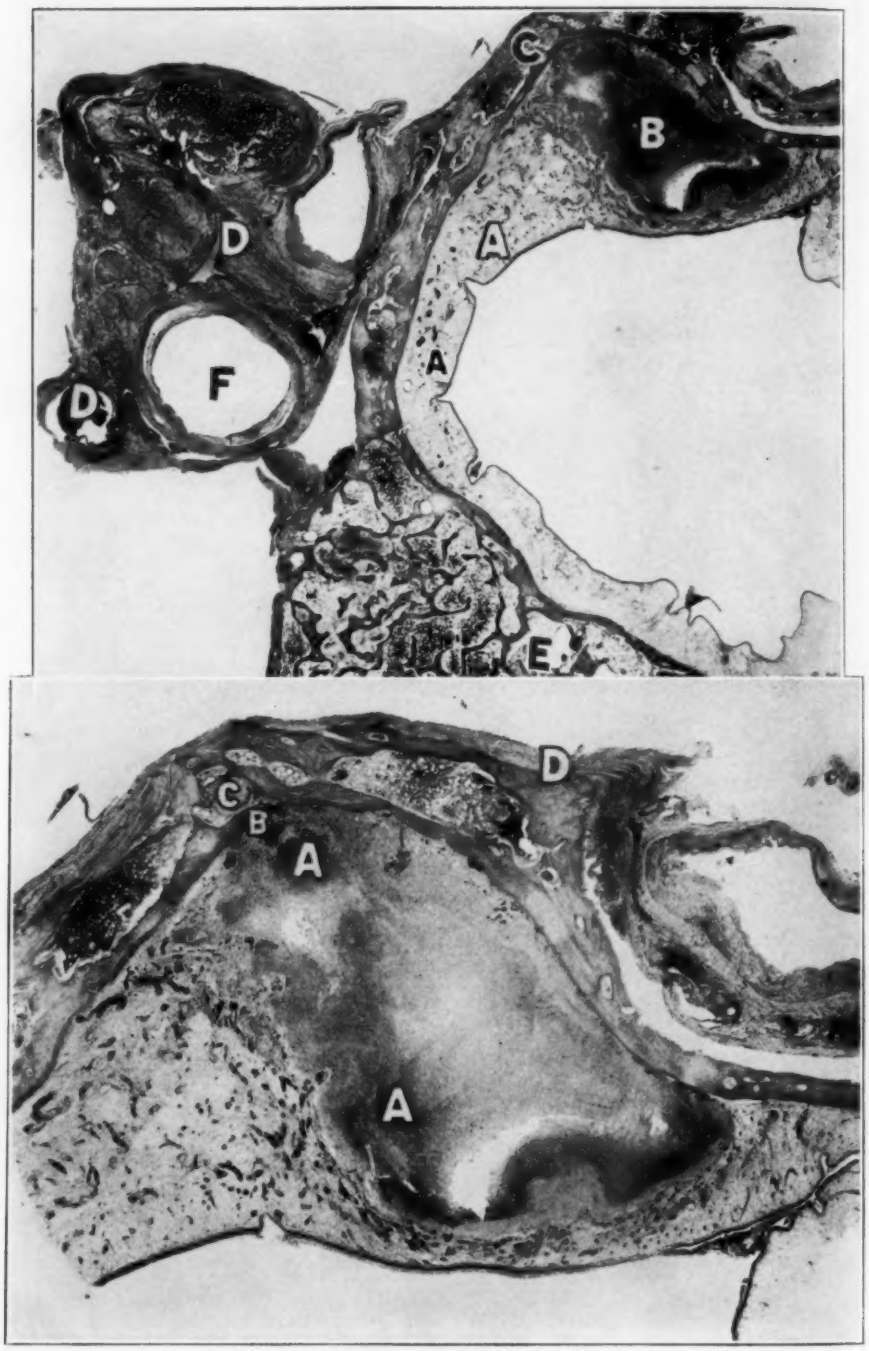

Fig. 9.—The upper portion, a section through the sphenoid sinus, shows a submucosal abscess and adjacent osteomyelitis of the posterior wall: *A*, mucosa; *B*, submucosal abscess; *C*, posterior wall of the sphenoid sinus; *D*, cavernous sinus; *E*, floor of the sphenoid sinus, and *F*, carotid artery. The lower portion is a higher power view of the submucosal abscess shown in the upper part: *A*, submucosal abscess; *B*, thrombosed vessel leading into the bone marrow; *C*, thrombophlebitis of the marrow vessel and osteomyelitis, and *D*, inflamed dura.

phlebitis in the venules or tributaries of the cavernous sinus to the sphenoid mucosa. Although the vessels are difficult to distinguish because of disruption due to the severe inflammation, they may be identified by elastic tissue stains. Such acute abscesses should not be confused with chronic submucosal abscesses, which are primary and not the result of retrograde thrombosis. Further differentiation is afforded

Fig. 10.—Section through the sphenoid sinus, showing a submucosal abscess associated with contiguous osteomyelitis: *A*, nasopharyngeal mucosa; *B*, floor of the sphenoid sinus; *C*, submucosal abscess; *D*, osteomyelitis.

by the fact that the primary abscess is evidently chronic and usually single whereas the thrombophlebitic abscesses are invariably multiple and acute.

Perineural Olfactory Lymph Spaces.—The perineural lymph channels surrounding the filaments of the olfactory nerve communicate with

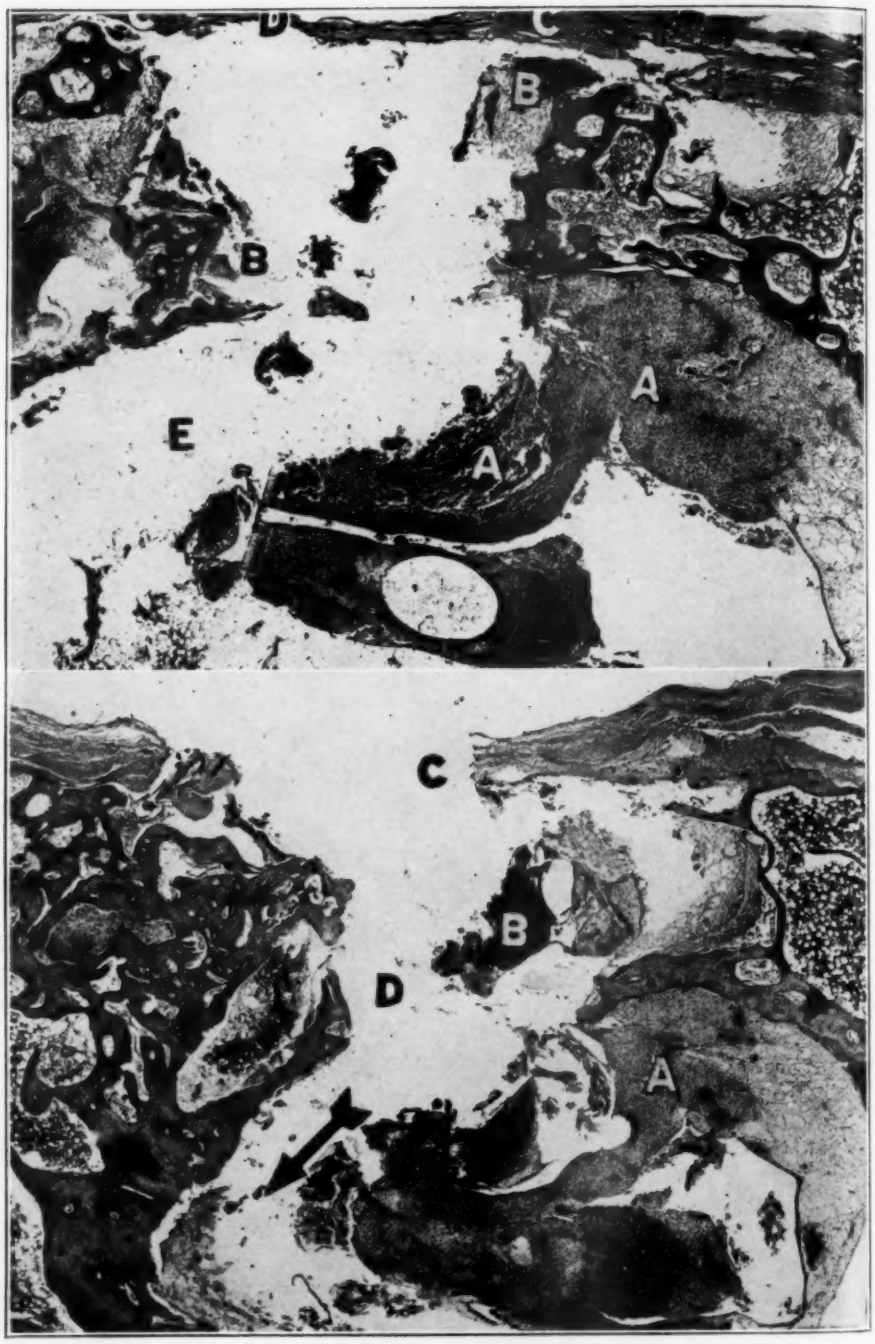

Fig. 11.—Higher power views of the section shown in figure 10. The upper part shows the section through the region of osteomyelitis: *A*, submucosal abscess; *B*, osteomyelitis with bone erosion; *C*, inflamed dura which has become thinned out at *D*, and *E*, irregular channel due to suppurative necrosis through which cerebrospinal fluid could escape. The lower view is of a section posterior to that pictured in the upper view, and shows the inflammatory defect of the dura: *A*, submucosal abscess; *B*, osteomyelitis; *C*, defect in the dura, probably due to rupture from increased intracranial tension, and *D*, irregular channel due to suppurative necrosis through which cerebrospinal fluid could escape.

the subarachnoid spaces endocranially and with the superficial network of lymphatics of the olfactory mucosa intranasally. This free lymphatic communication should appear to be an open avenue for the spread of infection to the meninges. However, experience has proved that function is more apparent than real. The perineural lymph channels are probably rapidly sealed off even when they are cleanly sectioned at operation. Except in rare instances, infection does not spread through these perineural sheaths to the subarachnoid spaces.

We have but 1 case in which we could be certain that the portal of entry was from the olfactory mucosa through the subarachnoid spaces via the perineural sheaths. In this instance there was acute inflammation together with marked leukocytic infiltration of the olfactory mucosa. Starting in the smaller filaments of the olfactory nerve there was perineural infiltration of polymorphonuclear leukocytes within the nerve sheath. This infiltration was continued around the larger nerve fibers and extended endocranially through the cribriform perforations to the subarachnoid spaces (fig. 12). The perineural spaces were dilated and freely invaded by leukocytes. In certain areas there was direct infiltration of the nerve fibers together with disintegration of nerve elements. Thrombosed vessels were nowhere to be seen (fig. 13, upper portion).

The origin of the perineural infection in the smaller radicles of the olfactory nerve in the mucosa below the cribriform plate is strongly stressed (fig. 12, lower portion). Without this evidence of the source of the inflammation we cannot attribute the spread to the perineural pathways. We have found perineural infiltration of the larger nerve fibers above the cribriform plate in suppurative meningitis not of sinus origin, viz., otitic infection and osteomyelitis of the basisphenoid bone.

Such perineural infection should be regarded as a frequent finding in suppurative meningitis and may be explained by the seepage or extension of the infiltration from the infected subarachnoid space into the open perineural sheaths, probably as the result of the higher cerebrospinal fluid pressure. In these instances in our series the infection was confined to the nerve sheaths above the cribriform plate, whereas the smaller filaments in the olfactory mucosa showed no such involvement.

We have an unusual instance of a dural polyp with a nerve filament forming its core extruded through the cribriform plate and entering the nasal cavity (fig. 13, lower portion). The patient had a cerebral neoplasm with markedly increased intracranial tension, which probably resulted in the herniation of this polyp (fig. 14, upper portion). Inflammation of the ulcerated dependent tip of the polyp in the olfactory sulcus spread to the subarachnoid space through the cribriform plate (fig. 14, lower portion).

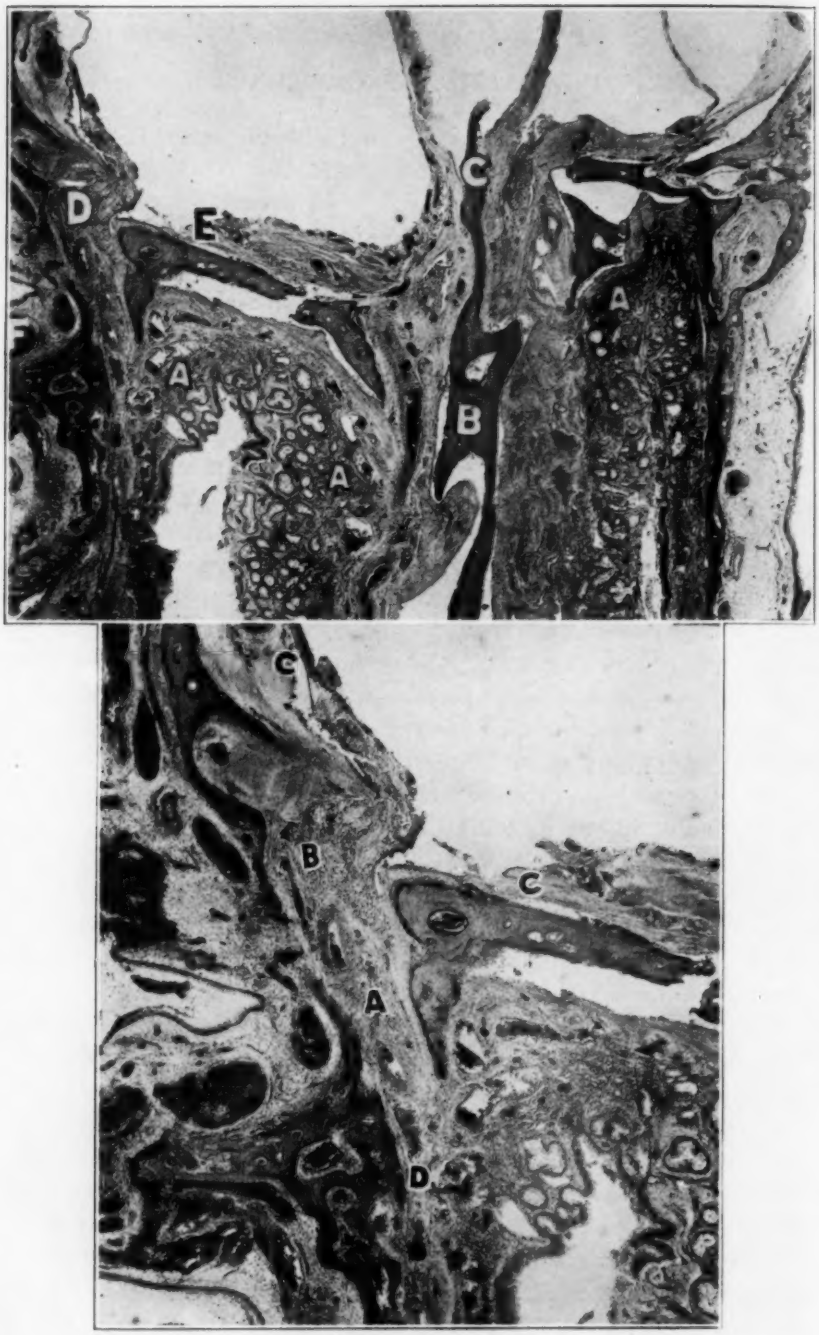

Fig. 12.—Section through the cribriform plate. The upper view shows olfactory fibers with perineural infiltration: *A*, inflamed olfactory mucosa; *B*, nasal septum; *C*, crista galli; *D*, nerve fiber with perineural infection passing through the cribriform perforation to the olfactory mucsoa; *E*, inflamed dura, and *F*, ethmoidal mucosa. The lower portion, a higher power view of the section at *D*, shows the perforating nerve fiber with infiltration of the perineural sheath: *A*, cribriform perforation; *B*, infiltration of the perineural sheath of the olfactory nerve; *C*, inflamed dura, and *D*, perineural infiltration of small nerve fibers in the olfactory mucosa.

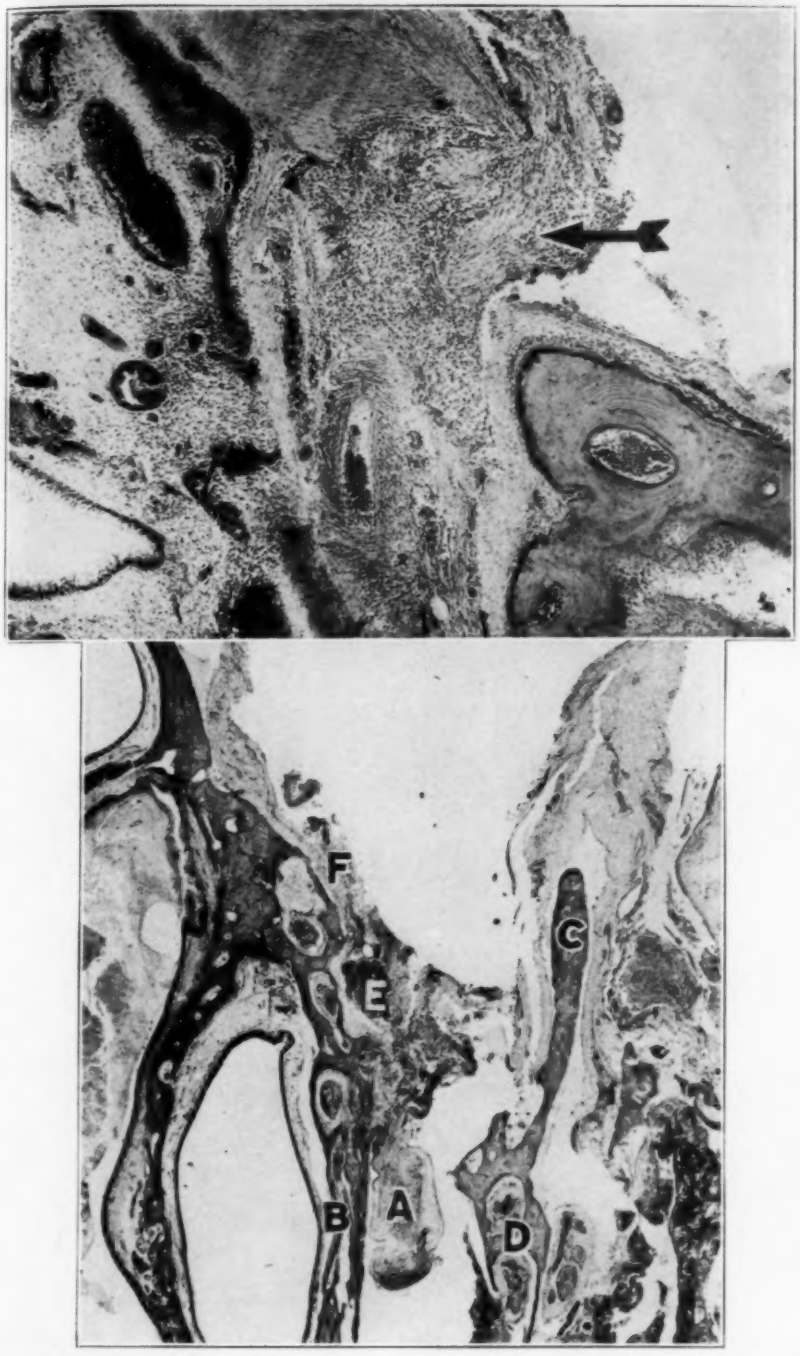

Fig. 13.—The upper portion is a higher power view of the section illustrated in the lower part of figure 12, and shows direct infiltration and fragmentation of the olfactory nerve by polymorphonuclear leukocytes. The lower portion, a section through the cribriform plate, shows a dural polyp in the right olfactory sulcus: *A*, dural polyp; *B*, right middle turbinate bone; *C*, crista galli; *D*, nasal septum; *E*, cribriform plate, and *F*, dura.

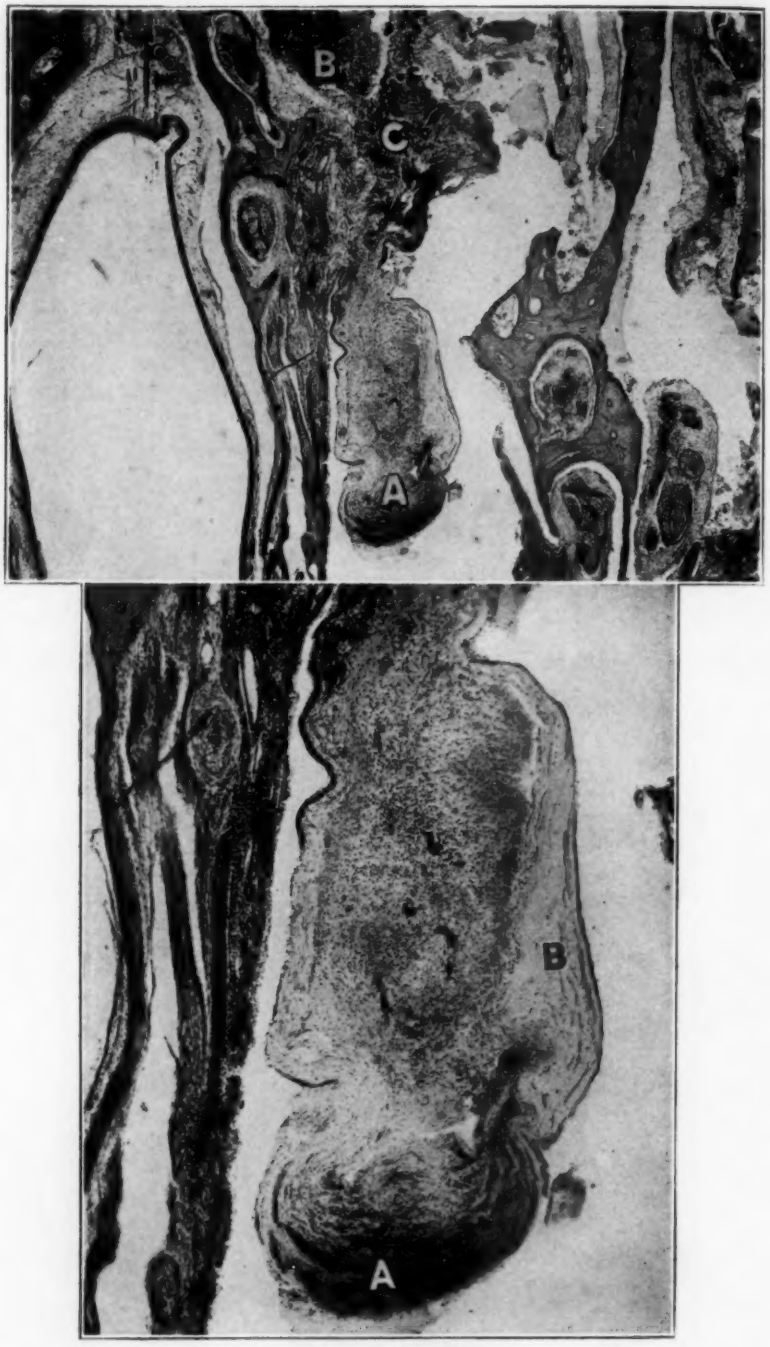

Fig. 14.—Additional views of the lesion shown in the lower portion of figure 13. The upper view, made at *A* under higher power, shows the dural polyp: *A*, dural polyp; *B*, inflamed dura over the cribriform plate, and *C*, perineural infiltration. The continuity of the dura with the polyp should be noted. The lower portion is a section through the dural polyp shown at higher power: *A*, ulcerated and inflamed tip of the polyp, through which infection occurred, and *B*, dural covering of the polyp due to herniation of the dura. The patient had cerebrospinal rhinorrhea.

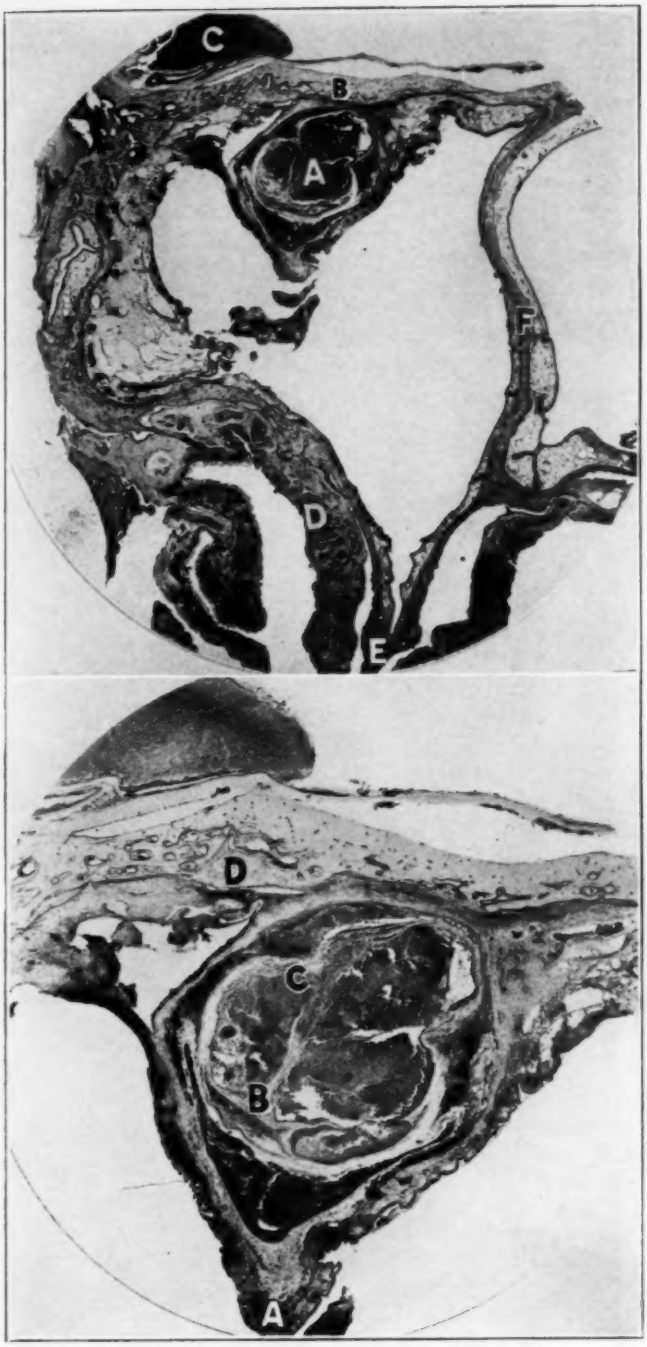

Fig. 15.—Section through the sphenoid sinus. The upper part shows a chronic submucosal abscess: *A*, submucosal abscess; *B*, posterior wall of the right sphenoid sinus; *C*, optic nerve; *D*, nasopharyngeal mucosa; *E*, rostrum, and *F*, intersphenoid septum. The lower part, a higher power view at the region of the submucosal abscess, shows: *A*, mucosa of the sphenoid sinus; *B*, fibrotic wall of the abscess; *C*, rupture of the capsule, and *D*, posterior wall of the sphenoid sinus, with perivascular infiltration.

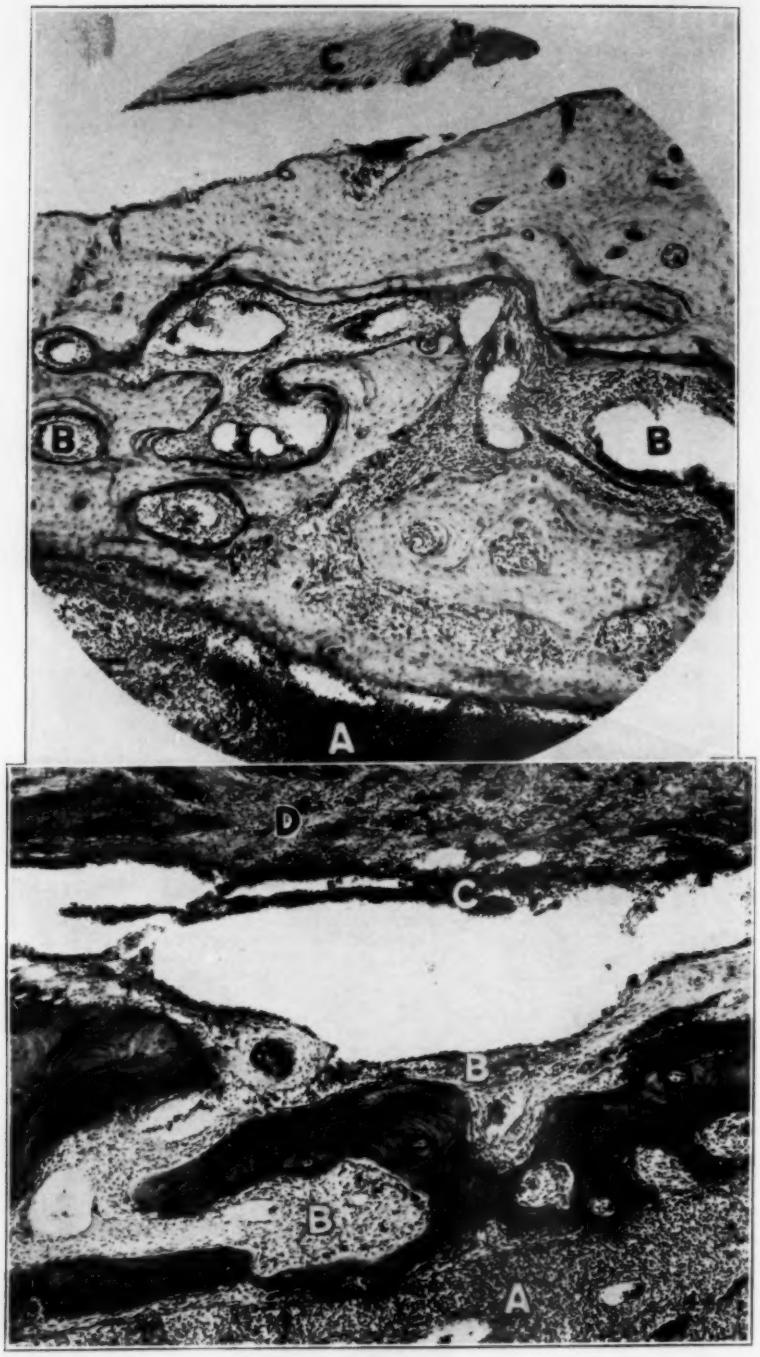

Fig. 16.—The same lesion as that shown in figure 15. The upper portion is a higher power view at the region of the posterior wall at *D*: *A*, base of the submucosal abscess; *B*, blood vessels, with perivascular infiltration of polymorphonuclear leukocytes, and *C*, dura. It is to be noted that the vessels are not thrombosed but that their perivascular spaces are infiltrated. The lower portion, a section through the posterior wall of the sphenoid sinus, shows communication of the perivascular spaces with the dura: *A*, base of the submucosal abscess; *B*, infiltrated perivascular spaces; *C*, epidural abscess, and *D*, inflamed dura.

Lymphatic Extension Through Perivascular Lymph Channels.—The perforating vessels which course from the sphenoid mucosa through the bone to the venous sinuses and dural vessels are surrounded by perivascular spaces which are probably lymph channels. In normal bone these perforating vessels are separated from their enclosing bony channels

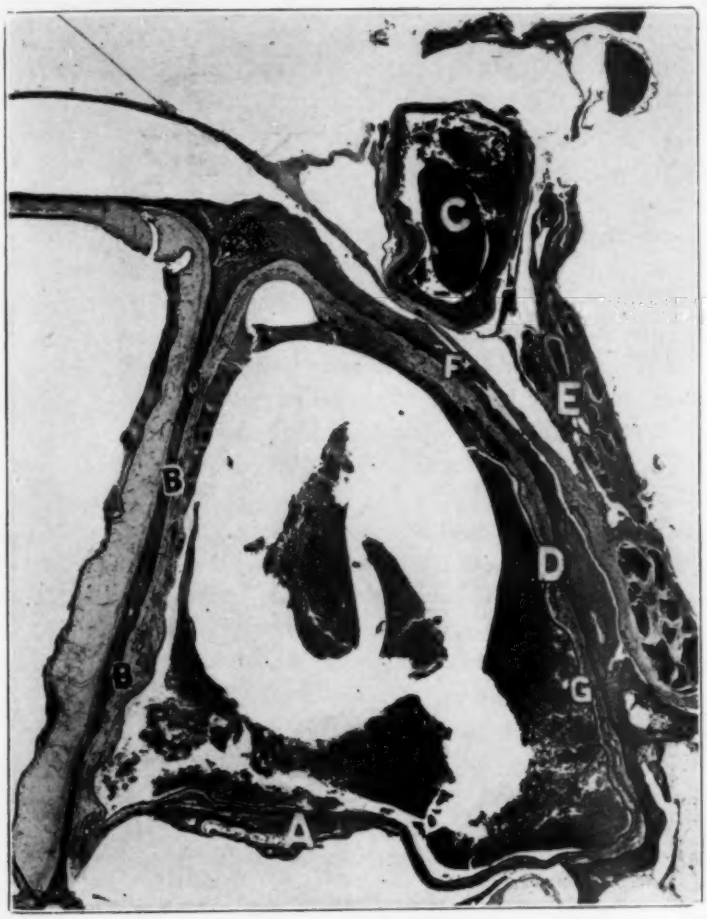

Fig. 17.—Section through a sphenoid sinus showing an empyema of the sinus: *A*, anterior wall of the sinus; *B*, intersphenoidal septum; *C*, carotid artery; *D*, lateral wall of the sphenoid sinus, and *E*, cavernous sinus. The perforating vessels at *F* and *G* are to be noted; these vessels communicate with the cavernous sinus directly.

by acellular spaces, which probably communicate with the lymph channels of the mucoperiosteum of the sphenoid sinus.

We have encountered spread of infection to the meninges by way of these perivascular lymph channels. The mucoperiosteum of the

sphenoid sinus contained a large chronic submucosal abscess with a distinct fibrotic capsule (fig. 15, upper portion). Near the posterior bony wall an acute infection had caused a break through the capsule and had invaded the subperiosteal space (fig. 15, lower portion). From this point the infection spread along the lymph channels surrounding the perforating vessels (fig. 16, upper part). The veins themselves were not thrombosed, but their perivascular spaces were infiltrated by polymorphonuclear leukocytes. Serial sections revealed that this infiltration continued along the blood vessels through the entire thickness of the bone up to the dura, at which site an extradural abscess was seen (fig. 16, lower part).

Vascular Spread by Venous Channels.—The vessels connecting the mucoperiosteum of the ethmoid and sphenoid sinuses with the cranial sinuses or their tributaries may become the site of thrombophlebitis. When the thrombosed venule extends through the os planum there may result thrombophlebitis of the ophthalmic vein and ultimately cavernous sinus phlebitis. Should the venule perforate the sphenoid wall, the cavernous sinus may become infected directly. Figures 17 and 18 illustrate a case of acute empyema of the sphenoid sinus, with thrombophlebitis of perforating vessels which directly communicate with and infect the cavernous sinus.

If the thrombosed vessel is part of a haversian system, first it may produce an osteomyelitis of the bone marrow and then infected veins may carry the infection to the cavernous sinus. An instance of this method of spread is illustrated in figure 19 (upper portion). There is seen intense acute inflammation of the mucoperiosteum of the sphenoid sinus. In the adjacent bone two dilated thrombosed venules may be observed. These can be traced to their origin in the mucoperiosteum and their termination in the marrow of the sphenoid bone, where osteomyelitis is present (fig. 19, lower portion). From this site the infection reached the cavernous sinus by diploic veins.

Spread by venous thrombosis is a relatively uncommon method of extension from the sphenoid and ethmoid sinuses. In acute frontal sinusitis associated with osteomyelitis, venous spread is much more frequent and probably the most usual method. Hajek [6] reported a case of meningitis following venous extension from an infected ethmoid sinus. Turner and Reynolds [8] found thrombophlebitis in 2 cases, in 1 in the sphenoid and in the other in the ethmoid sinus.

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8. Turner, A. L., and Reynolds, F. E.: A Study of the Paths of Infection to the Brain, Meninges, and Venous Blood Sinuses from Neighboring Pharyngeal Foci of Inflammation: Suppuration in Right Posterior Ethmoidal and Sphenoidal Sinuses; Orbital Abscess; Cavernous Sinus Thrombosis; Acute Leptomeningitis: Operation; Death; Autopsy, J. Laryng. & Otol. **41**:442 (July) 1926.

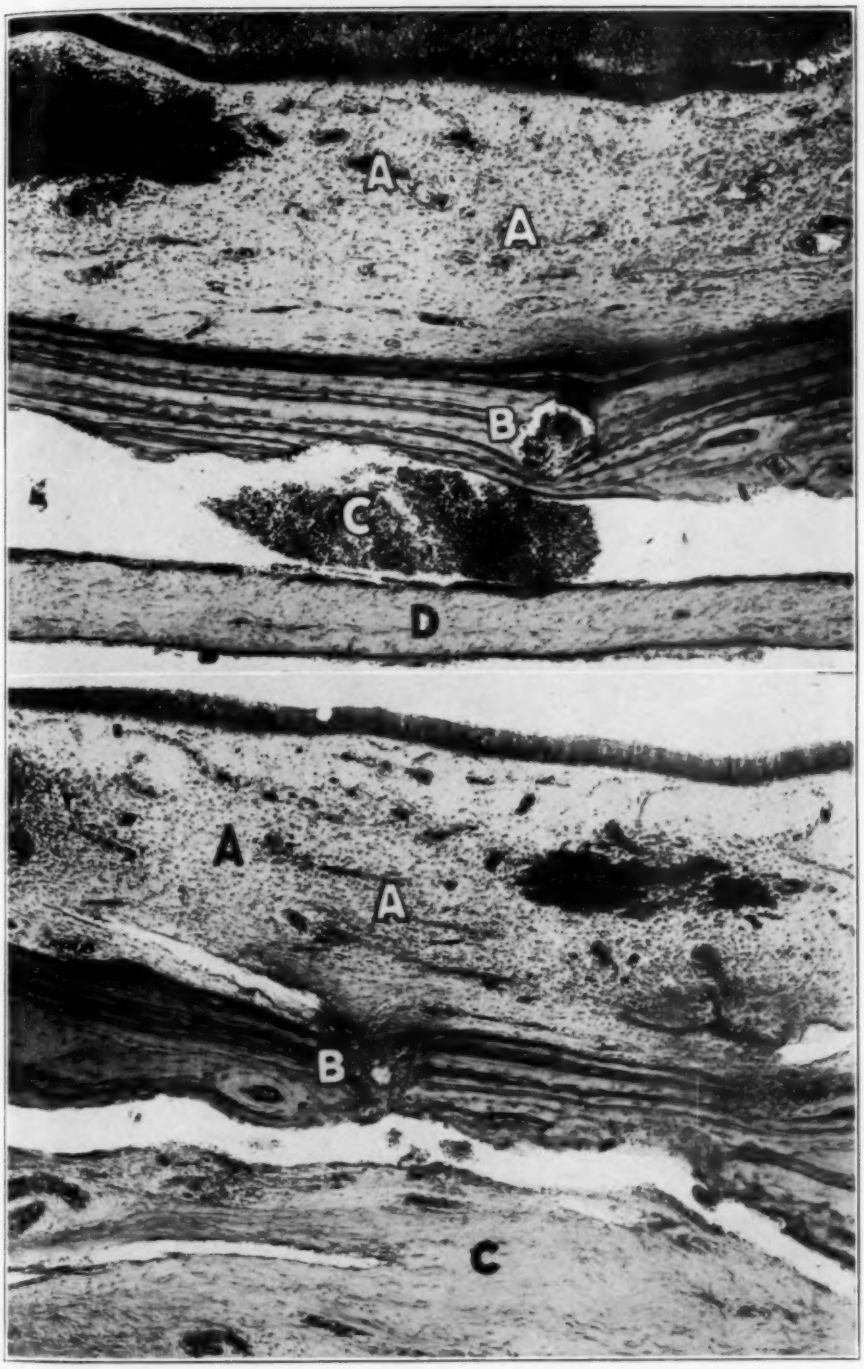

Fig. 18.—Higher power views of the section shown in the lower part of figure 16. The upper view, at *F*, shows a thrombosed venule in the lateral wall of the sphenoid sinus: *A*, inflamed mucosa of the sphenoid sinus; *B*, thrombosed vessel in the lateral wall; *C*, epidural abscess, and *D*, dural wall of the cavernous sinus. The lower view, at *G*, shows thrombophlebitis of a small venule communicating with the cavernous sinus: *A*, inflamed mucosa; *B*, perforating venule with phlebitis, and *C*, inflamed wall of the cavernous sinus.

Fig. 19.—Sections through the posterior wall of the sphenoid sinus. The upper view shows thrombophlebitis of communicating venules and osteomyelitis: *A*, intense inflammation of the mucosa of the sphenoid sinus; *B*, small thrombosed venule in the mucosa; *C*, thrombophlebitis of a vein leading to the bone marrow, and *D*, infiltrated dura. The lower view likewise shows thrombophlebitis of a venule producing osteomyelitis.

Direct Invasion of Meninges Through a Congenital Bony Defect.—Macroscopic congenital dehiscences are rare. Microscopic dehiscences are more common. We have encountered bony defects of the optic canal in which the condition was such that the nerve was in contact with the mucoperiosteum of the sphenoid sinus (fig. 20). Figure 21 (upper part) shows a congenital bony defect of the lateral wall of the sphenoid sinus. The internal carotid artery and cavernous sinus are in contact with the inflamed mucosa of the sphenoid sinuses. From this area invasion of

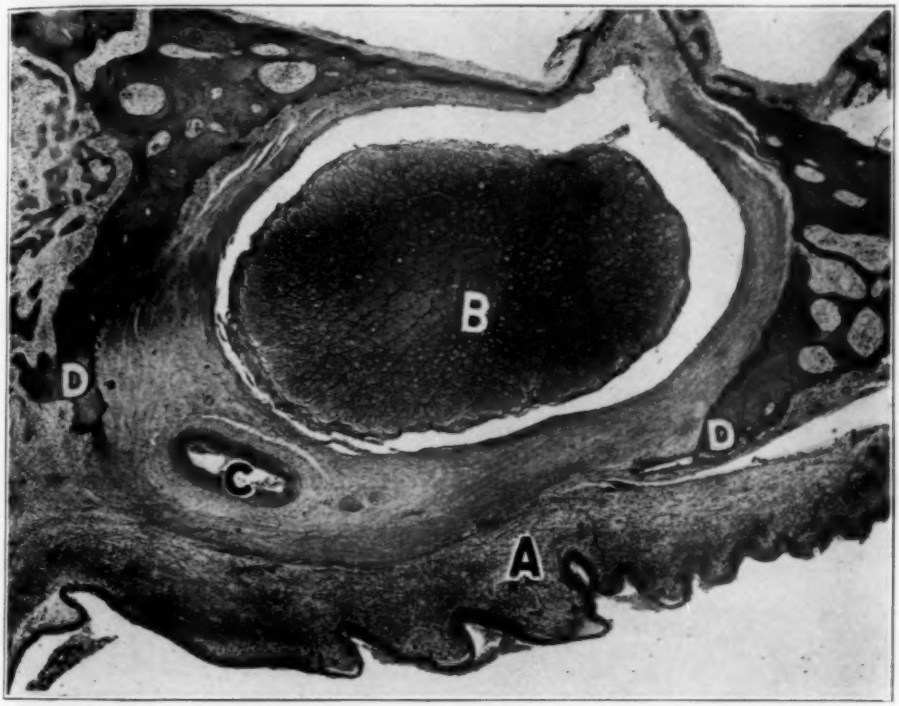

Fig. 20.—Section through the roof of a right sphenoid sinus, showing a bony defect of the optic canal: *A*, mucosa of the sphenoid sinus; *B*, optic nerve; *C*, ophthalmic artery, and *D*, limit of the bony wall.

the cavernous sinuses took place by direct spread through the bony defect (fig. 21, lower part).

Spread from a Persistent Cranial Pharyngeal Pouch.—The hypophysis originates from two different primordia: one the floor of the primary forebrain and the other the superior posterior surface of the ectoderm lining of the primitive mouth cavity. This ductlike communication of the hypophysis with the buccal cavity normally becomes completely obliterated. If obliteration is incomplete a cranial pharyngeal canal may persist, perforating the osseous cranial base and connecting with the nasopharynx.

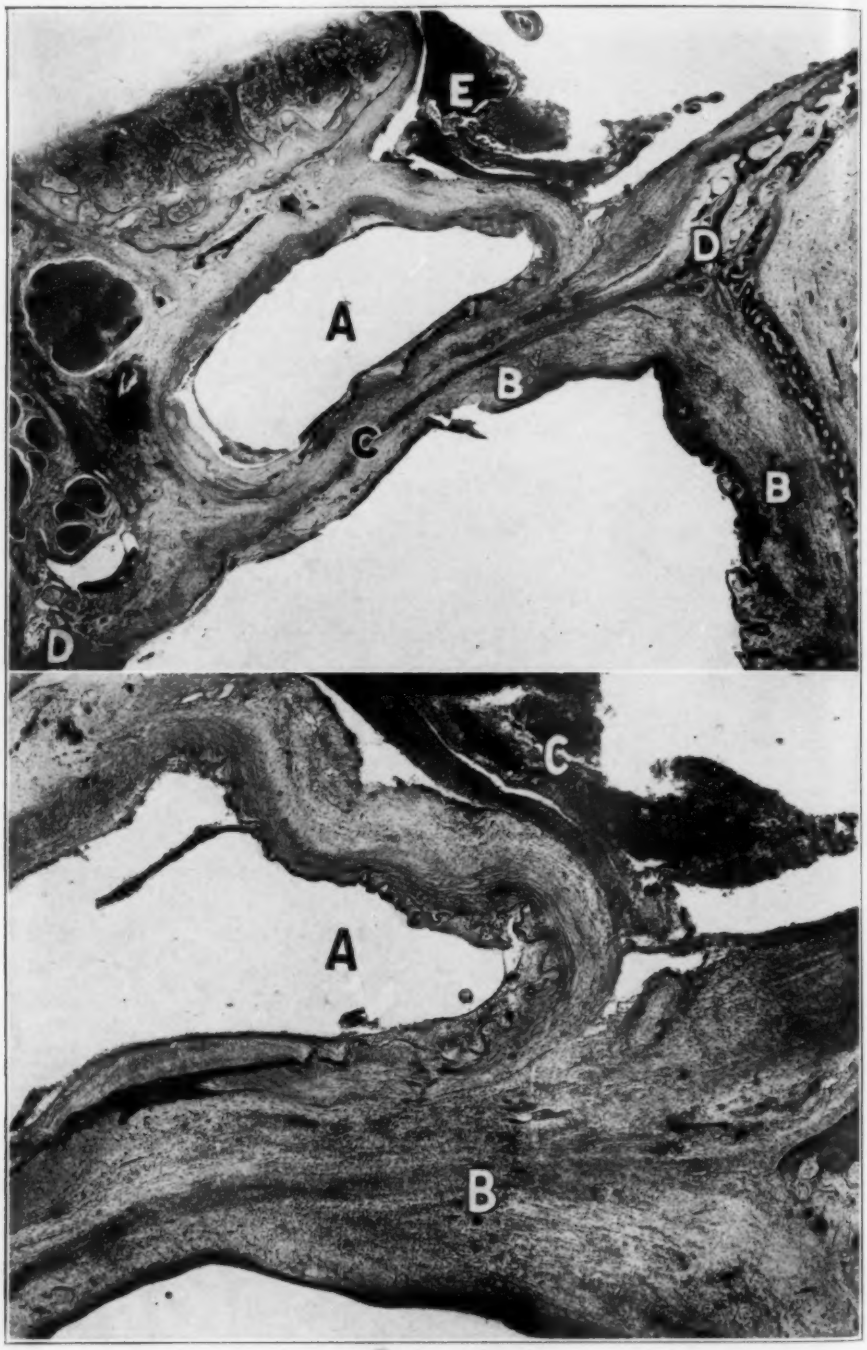

Fig. 21.—Section through a sphenoid sinus. The upper portion shows a congenital bony defect of the lateral wall of the sphenoid sinus. The carotid artery and cavernous sinus are in direct contact with the mucosa of the sphenoid sinus: *A*, carotid artery; *B*, mucosa of the sphenoid sinus; *C*, congenital defect of the bone; *D*, limit of the bony wall, and *E*, cavernous sinus. The lower view, made under higher power at the region of the bony dehiscence, shows infection of the cavernous sinus by contiguity with the inflamed mucosa of the sphenoid sinus: *A*, carotid artery; *B*, inflamed sphenoidal mucosa, and *C*, thrombosis of the cavernous sinus.

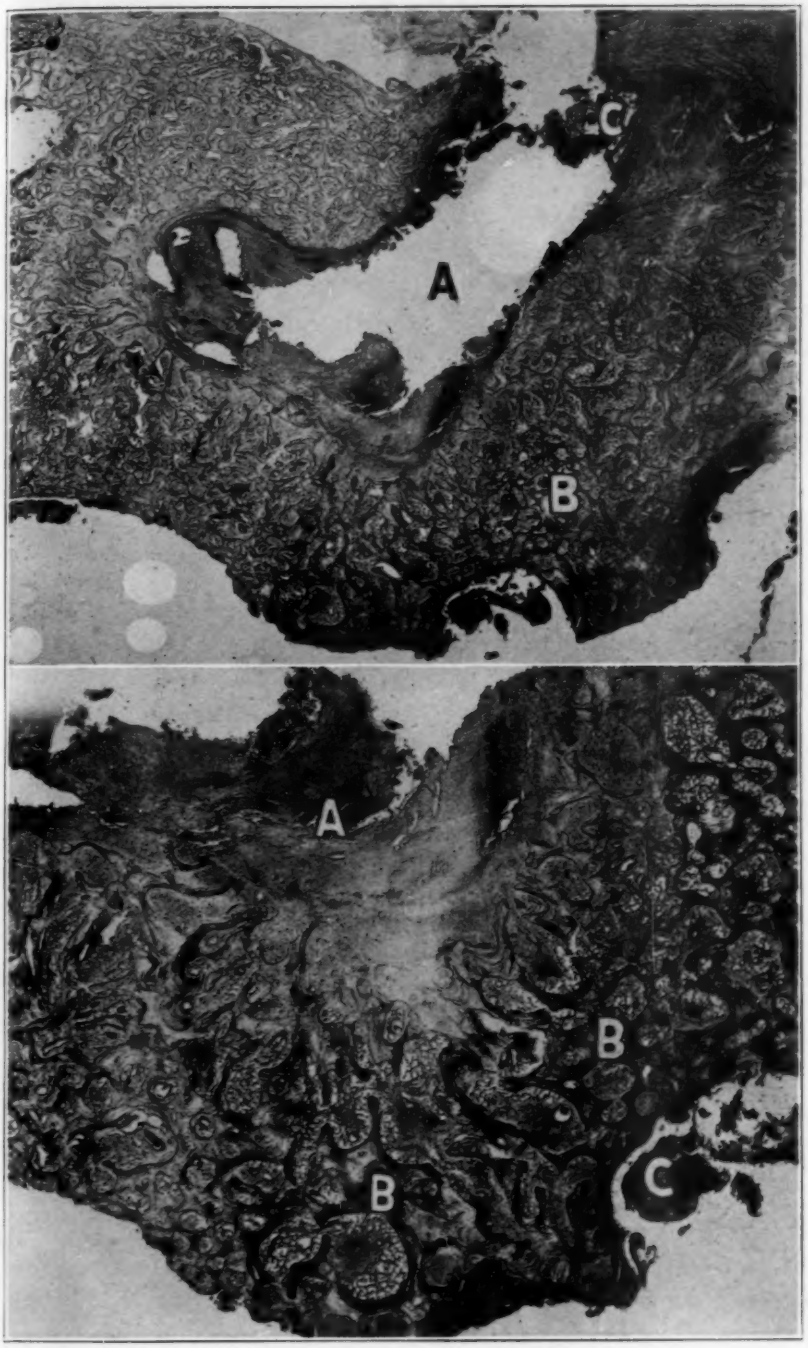

Fig. 22.—Section through the basisphenoid bone. The upper view shows a congenital craniopharyngeal pouch, extending into the basisphenoid bone from the sella turcica: *A*, craniopharyngeal pouch lined with inflamed dura; *B*, bony anterior wall of the nasopharynx, with osteomyelitis, and *C*, rupture of the inflamed dura with resultant meningitis. The lower view, made under higher power at *B*, shows intense inflammation of the dura lining the pouch: *A*, inflamed dura; *B*, acute osteomyelitis, and *C*, indentation into the nasopharynx, which probably represents the anlage of the anterior stalk.

In the case illustrated in figure 22 both sphenoid sinuses were absent. There was a persistent pouch in the basisphenoid bone, originating from the sella turcica and lined by a reflection of the dura from the middle fossa (fig. 22, upper portion). This pouch ended blindly near the nasopharynx in the region of the angle of the primitive stalk. The bone separating this pouch from the nasopharynx was the seat of an acute osteomyelitis. The infection spread from the nasopharynx through the marrow and then to the dura lining the pouch (fig. 22, lower portion).

SUMMARY

Numerous textbooks and special articles have tabulated various pathways and modes of intracranial involvement from sphenoid and ethmoid infections. With the exception of a few instances, no proof has ever been offered to substantiate the existence of assumed pathways.

We have been able to demonstrate the source of infection in cases of bacterial meningitis of so-called undetermined origin. Even after a complete postmortem examination at the Mount Sinai Hospital the source of the infection had in many instances remained unknown until serial sections of the sinuses revealed the primary infection. We stress this point because we have found that paranasal sinusitis may be the origin of intracranial complications even if gross examination reveals no abnormality. Macroscopic evidences of an infection were observed in but 3 of 50 sinus blocks studied.

A frequent finding in cases of meningitis resulting from inflammation of the sphenoid and ethmoid sinuses is the primary submucosal abscess. Although obvious microscopically, such abscesses are difficult to recognize at operation or postmortem examination. Because we have encountered them so often we believe that at operation it would be advisable to strip the mucosa of the sphenoid sinus in cases of meningeal irritation. It would appear that this is a more logical procedure than only the institution of adequate drainage.

We have illustrated spread to the meninges (1) by way of osteomyelitis and osteitis, (2) through the perineural olfactory lymph sheaths, (3) by lymphatic extension through perivascular lymph channels, (4) through vascular spread by venous channels, (5) by direct invasion of the meninges through congenital bony defects and (6) from a persistent cranial pharyngeal pouch.

Serial sections of the sinuses in all cases of bacterial meningitis should be made in an attempt to determine further the origins and methods of spread of the infection, with the hope of their ultimate prevention.

Dr. Paul Klemperer and Dr. Sadeo Otani, of the laboratories of Mount Sinai Hospital, assisted in the study.

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DEVELOPMENT OF THE OTIC CAPSULE

V. RESIDUAL CARTILAGES AND DEFECTIVE OSSIFICATION AND THEIR RELATION TO OTOSCLEROTIC FOCI

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The occurrence of residual cartilage or defective ossification in certain parts of the petrous bone in late fetal life or in early childhood has been recognized for some time. In the region of the fissula ante fenestram this defective development was found by Bast,[1] Anson and Wilson,[2] Anson and Martin [3] and Wilson [4] to occur in a large percentage of fetuses and young children. They suggested that this unstable condition in the region of the fissula ante fenestram may be one of the factors which initiate a late attempt at rebuilding of bone and which may result in sclerotic bone. Since otosclerotic bone usually occurs in this region and replaces the residual cartilages, the interpretation sounds plausible.

Another place where such residual cartilages occasionally occur is the region of the "fossula post fenestram." Bast [5] showed that residual cartilage or defective ossification in this location is rare and found it in only 5 per cent of the 93 petrous bones studied. Otosclerotic bone in this location is also rare.

Otosclerotic bone has been observed in other regions of the otic capsule. The early literature of otosclerosis will not be cited here because it is given in the two volumes on otosclerosis by the American Otological Society, published by Paul B. Hoeber, New York, 1929.

From the Department of Anatomy, the University of Wisconsin.

This study is supported in part by the Central Bureau of Research of the American Otological Society and the Wisconsin Alumni Research Foundation.

1. Bast, T. H.: (*a*) Osteogenesis of the Human Periotic Capsule, Arch. Otolaryng. **10**:459-471 (Nov.) 1929; (*b*) Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram, ibid. **18**:1-20 (July) 1933; (*c*) Development of the Otic Capsule: III. Fetal and Infantile Changes in the Fissular Region, ibid. **23**:509-525 (May) 1936.

2. Anson, B. J., and Wilson, J. G.: The Fissula Ante Fenestram in an Adult Human Ear, Anat. Rec. **56**:383-393 (July 25) 1933.

3. Anson, B. J., and Martin, J., Jr.: Fissula Ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303-323 (March) 1935.

4. Wilson, J. G.: Fissula Ante Fenestram and the Adjacent Tissue in the Human Otic Capsule, Acta oto-laryng. **22**:382-392, 1935.

5. Bast, T. H.: Development of the Otic Capsule: IV. The Fossula Post Fenestram, Arch. Otolaryng. **27**:402-412 (April) 1938.

A review of this literature gives the impression that in most cases in which otosclerotic bone was found in other parts of the otic capsule it occurred as an extension of the primary pathologic focus in the area of predilection, the region of the fissula ante fenestram. There are cases recorded, however, of multiple foci in various parts of the petrous bone which are not continuous with each other. Thus Brühl, in 1905, noted otosclerotic bone along the facial canal. In the region of the internal auditory meatus and the base of the cochlea such pathologic change in bone was noted by Habermann in 1903; Moller, 1904; Brühl, 1908; Siebenmann, 1911; Wolff, 1914, and Lange, 1921. Other workers, such as Mayer, 1915, and Brühl, 1913, found foci at the base or tip of the cochlear capsule. Siebenmann, in 1899 and 1911, Brühl in 1913 and Nager in 1926, observed them around the semicircular canals. In 1930 Hagens [6] also observed such pathologic bone around the semicircular canals and the cochlea. Recently Dr. Hagens permitted me to study his sections. The pathologic bony foci were of the branching type, apparently following blood channels. In the canal region they seemed to involve primarily the endochondral bone and not the surrounding bone.

Since residual cartilage or defective bone is commonly found in fetuses and young children in the region of the fissula ante fenestram, which is the site of predilection of otosclerotic bone, it is of interest to determine whether residual cartilages occur in these other areas where otosclerotic foci have been found. Last year Dr. J. Gordon Wilson suggested that all available fetal and infant petrous bones should be studied in an attempt to ascertain the location of all residual cartilages. This paper presents the results of such a study of my collection of fetal and infant petrous bones.

MATERIALS AND METHODS

More than 40,000 serial sections of 64 older fetal and 28 infant petrous bones were studied carefully. Reconstruction of typical petrous bones and photomicrographs of sections aided in accurate localization of the residual cartilages and areas of defective ossification.

OBSERVATIONS

Since the residual cartilages in the region of the fissula ante fenestram and the fossula post fenestram were described in earlier accounts, they will not be considered here. In the 92 petrous bones studied, residual cartilages or defective bone were found in four different regions besides the fissula ante fenestram and fossula post fenestram. The

6. Hagens, E. H.: Otosclerosis, Arch. Otolaryng. **12**:273-289 (Sept.) 1930.

four sites where such defects occur are, (1) the posteroinferior portion of the cochlear capsule, (2) in the basal portion of the styloid process, (3) at the petrosquamous suture and in the capsule beneath the suture, and (4) in the canal region.

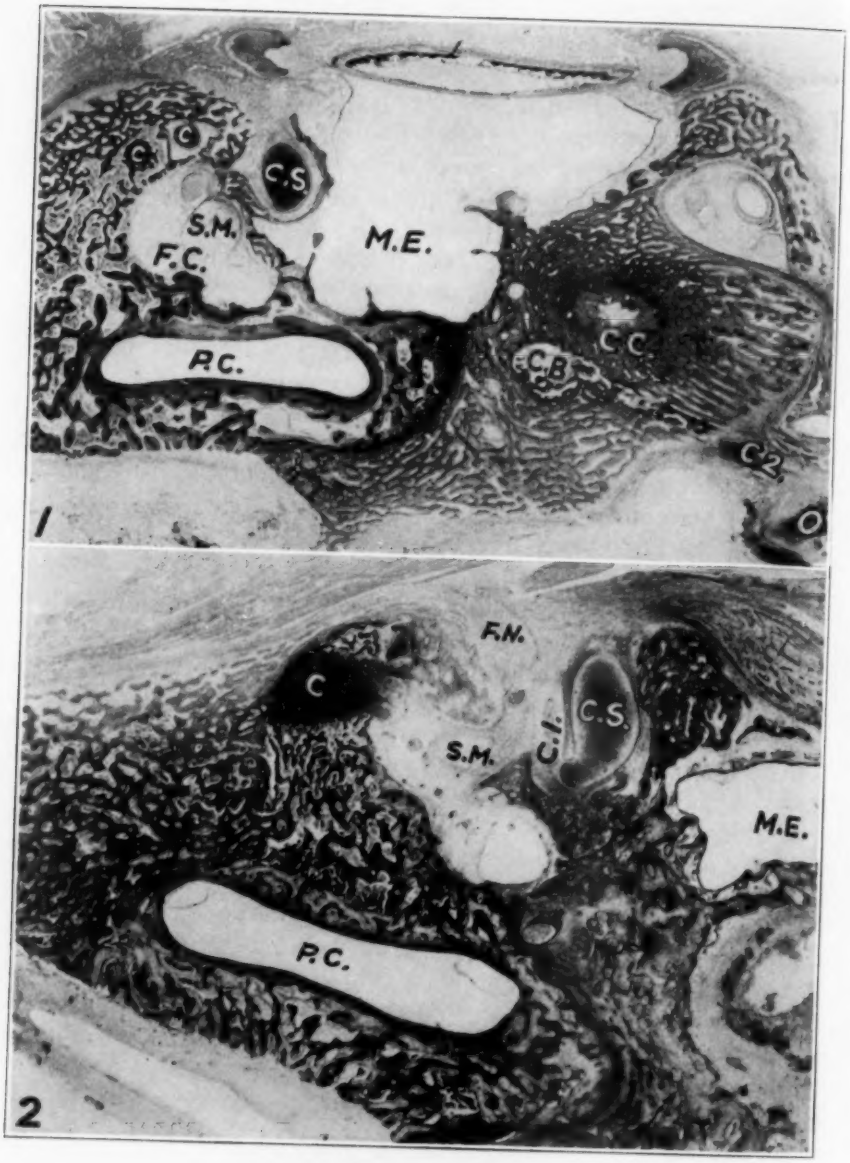
1. *Infracochlear Region.*—A mass of residual cartilage is found in the posteroinferior portion of the bony capsule of the cochlea below the internal auditory meatus in about 26 per cent of late fetal petrous bones. In a much larger percentage of late fetal petrous bones there occurs in this location a bar of endochondral bone surrounded by the perichondral bone of the otic capsule. In the 28 petrous bones of infants ranging in age from birth to 6 years, no such cartilage masses were seen but the bar of endochondral bone encased in the perichondral bone of the capsule is usually present. This bar of bone (fig. 1, *C. B.*) is of a different texture and composition from the rest of the capsule and can readily be distinguished from it.

In fetuses of two and a half to four months this residual cartilage or bar of endochondral bone is represented by a bar of cartilage which extends from the occipital cartilage to the base of the cartilaginous capsule of the cochlea. The base of this bar of cartilage lies in the position of the future bony spur of the occipital bone, which forms the medial wall of the jugular fossa. During the subsequent development the perichondral bone of the otic capsule surrounds the distal end of this cartilage. The cartilage later begins to ossify, but some of the cartilage may remain until birth. Some of the unossified cartilage was found in 20 per cent of full term fetuses but not in children.

Since this cartilage mass usually disappears at about the time of birth it is not likely to play a serious role in subsequent pathologic processes. The bone formed from this cartilage (fig. 1, *C. B.*) is, however, a much looser and poorer type of bone than the rest of the capsular bone and no doubt is subject to subsequent reorganization, which may or may not result in sclerotic bone. Nager and Fraser,[7] in figure 2 accompanying their paper, show otosclerotic bone in the cochlear capsule, part of which is in the position of the endochondral bone, *C. B.* of my figure 1. Whether this infracochlear area of residual cartilage or defective bone gives rise to osseous disease is a question which I cannot answer now. The position is such that any reorganization of it could readily extend into the region of the internal auditory meatus or the basilar part of the cochlear capsule.

2. *Styloid Region.*—The styloid bone and adjacent bony process develop as a rule from two distinct cartilages (fig. 2, *C. S.* and *C. I*). They remain cartilaginous for some time after the otic capsule is well ossified. The perichondral bone of the capsule partly envelops the base

7. Nager, F. R., and Fraser, J. S.: On Bone Formation in the Scala Tympani of Otosclerotics, J. Laryng. & Otol. **53**:173-180 (March) 1938.



Figures 1 and 2

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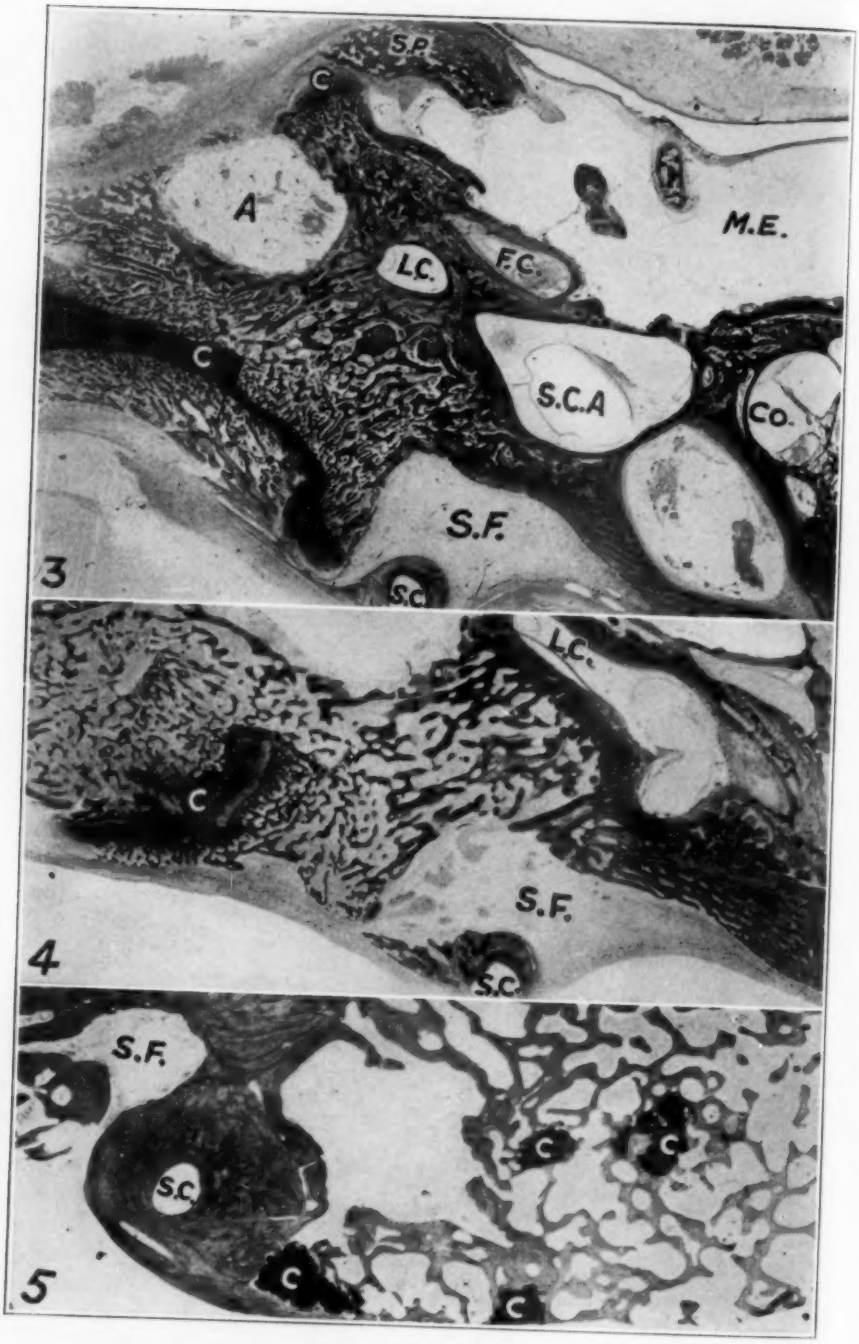
of the styloid cartilage. The tip of the styloid cartilage ossifies first, but its base, which later fuses with the petrous bone, remains cartilaginous until after birth. Ossification of the styloid cartilage as a rule is completed during the first three years of life, but in 1 case, that of a 6 year old child, a small mass of cartilage was still present. Late ossification of this styloid cartilage often results in a gnarly type of bone. The styloid bone is some distance from the otic capsule, so that in the preparation of older petrous bones for sectioning this region is usually cut away and thus any reorganization that may occur is not noted.

3. *Petrosquamous Suture Region.*—The cartilage of the primitive otic capsule as a rule stops its growth peripherally after ossification begins. This is not the case in the region lateral to the facial canal and anterior to the posterior and lateral canal, where the cartilage expansion keeps ahead of the ossifying process until it meets the expanding squamous plate. Normally this expanding cartilage is ossified at the time of birth except at the border, where it meets the squamous plate. Here a cartilaginous suture is found in 50 per cent of the infant bones examined (fig. 3, *C*). Ossification of the cartilage beneath the suture in a certain number of cases appears to be faulty. In this region residual cartilage masses were found in 33 per cent of full term fetuses and in 29 per cent of infant petrous bones. The letter *C* in figures 1 and 2 indicates such residual cartilages. In a few cases a center of poorly formed gnarly bone was found in place of the cartilage remnants. Since in most cases ossification is complete in this region before birth and since the percentage of residual cartilage is almost as great in infants up to 2 years of age as in full term fetuses, the condition suggests an abnormal delay in ossification. In older infants the occasional centers of poorly formed gnarly bone indicate a late attempt

EXPLANATION OF FIGURES 1 AND 2

Fig. 1.—Horizontal section through the base of the cochlea and the posterior semicircular canal of the left petrous bone of a human fetus (embryo 60, 305 mm. crown-rump length, approximate age 33 plus weeks). *C.B.* indicates the ossified bar of infracochlear cartilage; *C.2.*, the cartilaginous extension of the bar of cartilage; *O*, the occipital bone; *C.C.*, the cochlear capsule; *P.C.*, the posterior canal; *M.E.*, the middle ear; *F.C.*, the facial canal; *S.M.*, the stapedius muscle; *C.S.*, the styloid cartilage, and *C*, two masses of cartilage in the bone, extending deep to the petrosquamous suture.

Fig. 2.—Section similar to that in figure 1 (embryo 68, left, 310 mm. crown-rump length, approximate age 34 weeks). Two cartilages in the styloid region are shown, at *C.S.* and *C.1.* The cartilage mass deep to the petrosquamous suture is shown at *C*. *F.N.* indicates the facial nerve; *S.M.*, the stapedius muscle; *P.C.*, the posterior canal, and *M.E.*, the middle ear.



Figures 3, 4 and 5

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at conversion of this cartilage into bone. I know of no reported cases of otosclerotic bone in this region. It may be that, in view of the fact that this region forms the floor of the tympanic opening of the antrum, the area is resorbed as the opening of the antrum is enlarged with age.

4. *Canal Region.*—In 1932 I [8] described the development of the otic capsule in the canal region. I showed that much of the original cartilage is removed and replaced by an invasion of vascular connective tissue from the subarcuate fossa before ossification. The remaining cartilage and the vascular connective tissue are then gradually replaced by bone. In the aforementioned report the impression is given that in the last month of fetal life the entire capsule is ossified and that the connective tissue in the subarcuate fossa becomes more or less replaced by bone.

Since 1932 I have collected a larger series of petrous bones of the late fetal period and of early childhood. A study of this series revealed that the otic capsule is largely ossified at birth but that isolated spots of cartilage do persist in the bony capsule of the canal region in about 65 per cent of late fetal petrous bones or in those infants up to 3 years of age. In a pair of 6 year old petrous bones isolated patches of poorly formed gnarly bone occurred in this region, which indicates a late attempt of ossification of such residual cartilage masses.

The masses of residual cartilages usually occur lateral to the subarcuate fossa. There is, however, no specific place where they may lie. They frequently lie far laterally, at the lateral border of the petrous bone, sometimes they lie near the dural margin, at the lateral margin of the subarcuate connective tissue or extending into it, or in rare

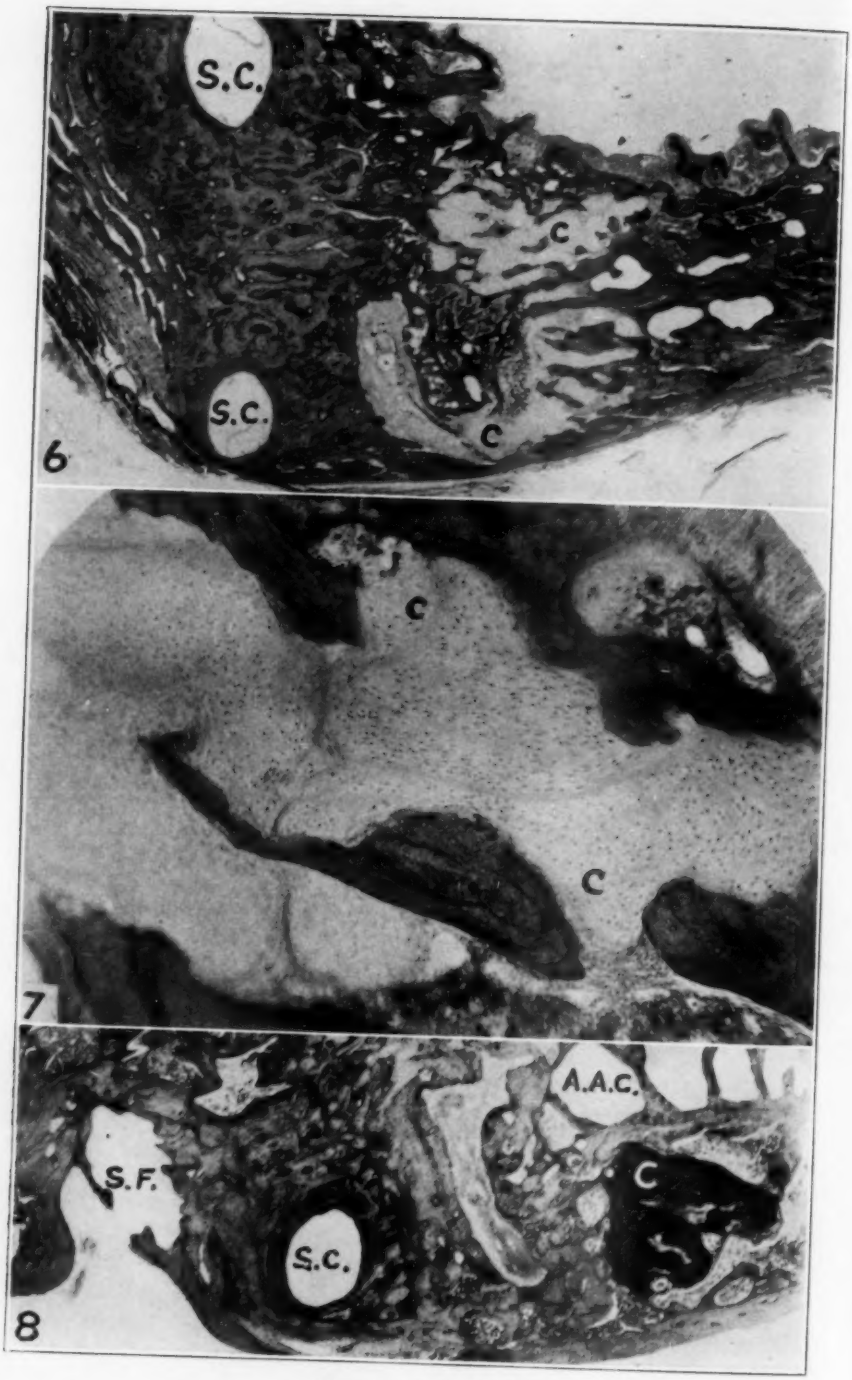
8. Bast, T. H.: Development of the Otic Capsule: I. Resorption of Cartilage in the Canal Portion of the Otic Capsule, Arch. Otolaryng. **16**:19-38 (July) 1932.

EXPLANATION OF FIGURES 3, 4 AND 5

Fig. 3.—Section through the same petrous bone shown in figure 2, but at a higher level. It shows cartilage, *C*, at the petrosquamous suture and another cartilage, *C*, in the canal region lateral to the superior canal, *S.C.*, and the subarcuate fossa, *S.F.* *A* indicates the antrum; *L.C.*, the lateral canal; *S.C.A.*, the ampulla of the superior canal; *F.C.*, the facial canal; *M.E.*, the middle ear; *S.P.*, the squamous plate of bone, and *Co.*, the cochlea.

Fig. 4.—Section through the petrous bone of another human fetus (embryo 47, left, size 315 mm. crown-rump length, approximate age 34 plus weeks). Massive residual cartilage is shown, lateral to the subarcuate fossa, *S.F.*, and the superior canal, *S.C.* The lateral canal is indicated by *L.C.*

Fig. 5.—Section through the ear of an infant (baby 103, right, age 1 year, 10 months and 20 days). Four masses of cartilage, *C*, are shown lateral to the subarcuate fossa, *S.F.*, and the superior canal, *S.C.*



Figures 6, 7 and 8

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instances they may occur in the arch of the lateral canal or even lateral to the posterior canal.

Some of these cartilages are surrounded by bone, while others lie at the margin of the subarcuate connective tissue and may extend into it. It has not been possible to determine whether they are remnants of the original capsular cartilage or whether they are the products of chondrification of some of the vascular connective tissue which became pinched off by the ossification process. Such pinching off of connective tissue processes by the growing bone was pictured in my previous paper.[8] When such masses of connective tissue become surrounded by bone their blood supply is diminished, and if they are of any size they may change to cartilage prior to ossification. Whether this actually occurs could not be verified from my sections since no direct transformation of connective tissue into cartilage was observed. The only evidence in favor of this secondary cartilage formation is the fact that about 10 per cent of the cartilages show signs of growth. They are either invading bone marrow spaces or the subarcuate connective tissue. In figures 6 and 7 such a growing cartilage is shown pushing into bone marrow spaces. In figure 9 the cartilage is extending into the connective tissue of the subarcuate fossa. In figures 10 and 11 portions of the cartilage shown in figure 9 are seen invading the bone marrow spaces.

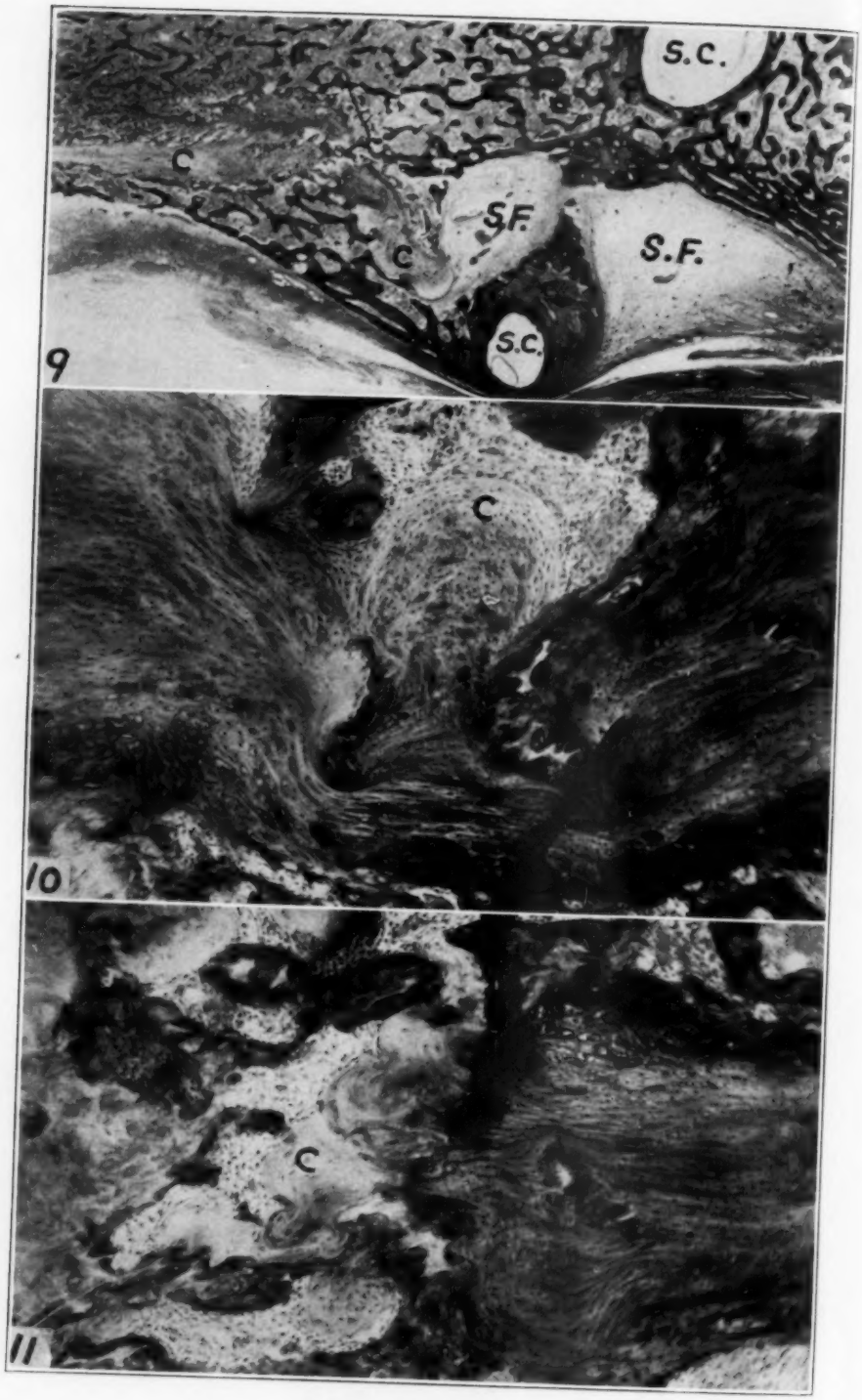
Such neoplastic cartilage, as already stated was noted in only 10 per cent of the cases, and what it may lead to cannot be determined now. In the other 90 per cent of cases the quiescent type of cartilage is observed. In these the cartilage is of various size, shape and contents. A few of these types are shown in figures 3, 4, 5 and 8. They usually lie lateral to the subarcuate fossa and may be single large cartilages as shown in figure 3 or multiple as in figure 5. The oldest child in whom such a cartilage was noted was 5 years old (fig. 8). Whether these cartilages which appear quiescent are all remnants of the original capsular cartilage or whether they were regenerating cartilages at one time, cannot be determined from my sections.

EXPLANATION OF FIGURES 6, 7 AND 8

Fig. 6.—Section through the ear of a human embryo at term (baby 130, right, at term), showing neoplastic cartilage at *C*, lateral to the superior canal, *S.C.*

Fig. 7.—Magnified view of part of the neoplastic cartilage shown in figure 6. This figure shows how the cartilage is pushing into the bone marrow spaces.

Fig. 8.—Section through the ear of a child (baby 118, right, age 5 years), showinto a large cartilage mass, *C*, lateral to the superior canal, *S.C.*, and the subarcuate fossa, *S.F.* *A.A.C.* indicates the antral air cells. This is the oldest subject in whom cartilage was noted in the canal region.



Figures 9, 10 and 11

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As far as I can gather from the literature, otosclerotic foci in the canal region occur as a rule in the endochondral bony capsule immediately surrounding the semicircular canals. This is also the condition in Dr. Hagen's subject, which I have examined. Since residual cartilages in the canal region lie outside the endochondral bone, either in the arch of the superior canal or often far lateral to it, there is no apparent relationship between them and the otosclerotic foci cited in the literature.

The only exception is that of a case reported by Siebenmann.[9] He described the focus as lying lateral to the superior canal and extending from the subarcuate fossa laterally beyond the limits of his sections. This location corresponds closely to that of observed residual cartilages. The statement of Siebenmann that the focus extended beyond the lateral limit of his section raises a pertinent question. How carefully have otosclerotic foci been sought for in the *canal region* of petrous bones? I do not know, but the pictures in the literature seldom show the canal region lateral to the vestibule. Those otosclerotic foci of the canals which are shown are at the vestibular end of the canals. How about the rest of the canal region? Is it not possible that the foci noted on the canals are extensions from some other, a primary, focus?

Residual cartilages must be considered as defective areas of ossification. It is to be expected that at some period of growth such areas are to be repaired. In a number of cases observed such repair results in a poorly formed gnarly bone. What happens to such unstable spots later? It would seem important that investigators who have occasion to study petrous bones of older children or otosclerotic petrous bones should take into account those regions where residual cartilages or defective bone have been noted in fetuses and young children.

SUMMARY

Residual cartilage or defective bone has been noted in the following regions of the petrous bone:

1. The region of the fissula ante fenestram, commonly.
2. The region of the fossula post fenestram, rarely.

9. Siebenmann, F.: Multiple Spongiosierung der Labyrinthkapsel, Ztschr. f. Ohrenh. **34**:356, 1899.

EXPLANATION OF FIGURES 9, 10 AND 11

Fig. 9.—Section through the ear of an infant (baby 124, left, age 4 days), showing the growing type of cartilage, *C*, lateral to the subarcuate fossa, *S.F.*, and the superior canal, *S.C.* The cartilage is budding into the subarcuate connective tissue.

Figs. 10 and 11.—Enlarged views of portions of the cartilage seen in figure 9. The growing cartilage buds, *C*, are shown, pushing into the bone marrow.

3. The infracochlear region.

4. The base of the styloid bone.

5. The region of the petrosquamous suture and in the capsule beneath the suture.

6. The region of the semicircular canals.

The later attempt at repair of such bony defects requires further study to determine whether they are related to the formation of otosclerotic foci.

Case Reports

HEMANGIOMA OF THE LARYNX IN INFANTS

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Hemangioma of the larynx is an uncommon condition. Irwin Moore [1] in 1923 was able to find only 76 cases in the literature; these included his own reported at that time. Hückel [2] in 1928, in a study of the records of a series of 26,999 patients admitted to the Göttinger Institute, reported the occurrence of 123 benign tumors of the larynx; of these only 4 were hemangiomas. New and Erich,[3] in reviewing the cases observed at the Mayo Clinic during the last thirty years, found 722 benign tumors of the larynx; 26 of these growths were hemangiomas.

Laryngeal hemangiomas were first classified by Sweetser [4] in 1921, into the adult and the infantile type. The former type occurs usually on or above the vocal cords, and the latter, subglottically. The hemangioma in the case we are about to report falls into the infantile group. We have been able to find only 5 such cases recorded. Two of these, the case of Phillips and Ruh [5] and that of Sweetser, were histologically proved to be instances of true hemangiomas, while the other 3, those of New and Clark [6] and Levbarg,[7] were not. To these 5 we add 2 cases of hemangioma of the upper part of the trachea, recently recorded by Hofmann.[8]

The table briefly summarizes the cases reported in the literature.

REPORT OF A CASE

C. D., a white boy aged 14 weeks, was admitted to the Jefferson Hospital at 4 a. m. on July 4, 1939, with a history of having been well until three weeks before, when the parents first noticed that the cry was hoarse and breathing was noisy at

From the Departments of Laryngology and Bronchoscopy and from the Clinical Laboratories of Jefferson Hospital.

1. Moore, I.: Further Remarks on Angiomata of the Larynx, J. Laryng. & Otol. **38**:57 (Feb.) 1923.

2. Hückel, R.: Hyperplastic Capillary Hemangioma of the Larynx, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **27**:193 (June) 1928.

3. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx: A Study of Seven Hundred and Twenty-Two Cases, Arch. Otolaryng. **28**:841 (Dec.) 1938.

4. Sweetser, T. H.: Hemangioma of the Larynx, Laryngoscope **31**:797 (Oct.) 1921.

5. Phillips, J., and Ruh, H. O.: Angioma of the Larynx, Especially Its Relation to Chronic Laryngitis, Am. J. Dis. Child. **5**:123 (Feb.) 1913.

6. New, G. B., and Clark, C. M.: Angiomas of the Larynx: Report of Three Cases, Ann. Otol., Rhin. & Laryng. **28**:1025 (Dec.) 1919.

7. Levbarg, J. J.: Intubation for Angioma of the Larynx, Laryngoscope **28**:867 (Dec.) 1918.

8. Hofmann, L.: Tracheal Hemangioma with Report of Two Cases, Ztschr. f. Hals-, Nasen- u. Ohrenh. **44**:435, 1938.

times. There was no fever. The child's condition remained unchanged until two weeks later (June 28), when there suddenly occurred dyspnea associated with stridor, croupy cough and inspiratory indrawing at the suprasternal notch and epigastrium. This lasted several hours and gradually subsided. Two days later (June 30) there was a similar attack of respiratory difficulty, which again lasted a few hours and slowly disappeared. At times the breathing seemed almost normal; hence nothing was done. On July 2, however, the stridor, dyspnea and indrawing reappeared and became progressively worse. The infant became restless and would not sleep or take his feedings. On the night of July 3 the family consulted a physician, who referred the patient to the bronchoscopic clinic.

The family history and birth history were entirely normal. The infant had had no previous illness. Gain in weight had been normal. There were no other children.

Examination.—Physical examination on admission revealed a fairly well nourished infant with signs of obstructive dyspnea, a hoarse cry, inspiratory stridor and marked indrawing at the suprasternal notch, the supraclavicular fossae and the epigastrium. There was cyanosis about the lips and nail beds, and a curious purplish mottling of the skin over the abdomen was seen. Respirations were extremely rapid. The pupils were equally dilated. The nose, mouth and throat were normal except for slight redness of the pharynx. No ulceration or membrane was noted. Examination of the chest revealed a few moist rales at both bases and distant breath sounds throughout, as well as extremely shallow excursions. There were no localized areas of dulness, but the entire chest was hyporesonant to percussion.

Treatment and Course.—Immediately after examination a low tracheotomy through the third ring of the trachea was performed, and a no. 0 tube was inserted. After the tracheotomy a moderate quantity of thick secretion was coughed up through the tracheal fistula, the indrawing was relieved immediately and the baby fell asleep. The respiratory rate fell to 40 per minute. The rectal temperature was 99 F. The improvement was only temporary, however, and it was necessary to aspirate secretion through the tracheotomic fistula at frequent intervals. There were frequent periods of cyanosis which could not be explained, as the airway seemed free. A no. 1 tube was substituted for the no. 0, and the patient was placed in an oxygen tent. Fluids were given by hypodermoclysis and through a nasal catheter. On this regimen the patient's condition remained fairly good throughout the day, but toward evening the dyspnea gradually increased and breath sounds became more distant. Palsy due to paralysis of the left sixth nerve was noted. The temperature gradually rose to 107.5 F. (rectal); the respiratory rate increased to 90 per minute, and death occurred at 1:30 a. m. on the following morning, twenty-two and a half hours after admission.

Necropsy.—Macroscopic Observations: The body was that of a well developed, well nourished white male infant approximately 3 months of age. On external examination the only observations of importance were a recent low tracheotomy incision and the absence of any cutaneous, buccal or pharyngeal hemangiomas. The thymus grossly appeared normal.

Just below the left vocal cord on the lateral aspect of the larynx there was a moderately firm, fusiform, submucosal tumor mass. It overlay part of the thyroid and cricoid cartilages and measured approximately 2 cm. in length, 1 cm. in width and 0.5 cm. in thickness. It almost entirely obliterated the larygeal air passage. The covering mucosa appeared intact. Section showed it to be bluish red, homogeneous throughout and sharply demarcated from both the mucosa and the

Author	Sex of Patient	Age in Months	Outstanding Symptoms	Duration	Remission	Location of Tumor	Angiomas Elsewhere	Method of Diagnosis	Treatment	End Result	Associated Pathologic Condition
Subglottic Hemangioma											
Phillips and Ruh	M	9	Dyspnea; stridor; indrawing	3 mo.	Yes (1)	Subglottic and in the upper part of the trachea	No	Autopsy	None	Asphyxia; death after 24 hr.	Lobar pneumonia
New and Clark....	F	9	Dyspnea; stridor	6 mo.	Not stated	Subglottic	No	History and laryngoscopy (no biopsy)	Radium, 250 mg. hr.	Improved	None
Sweetser.....	M	2	Dyspnea; cyanosis; indrawing	First attack at age of 9 days	Yes (1)	Subglottic	No	Autopsy	Intubation, unsuccessful	Asphyxia; death same day	None
Hemangioma of the Upper Part of the Trachea											
Hofmann.....	M	2	Dyspnea; stridor; indrawing	Since birth	Yes (1)	Upper part of the trachea	No	Laryngoscopy and autopsy	Tracheotomy; removal by tracheolaryngofissure	Pneumonia; death 1 mo. postoperatively	Pneumonia
Hofmann.....	M	2	Dyspnea; stridor; indrawing	Since birth	Yes (4)	Upper part of the trachea	Yes	Laryngoscopy confirmed by autopsy	Roentgen therapy, total 400 r; followed by tracheotomy	Marked improvement 16 days; death 24 hr. later	Bronchitis; pneumonia
Doubtful Cases											
New and Clark....	F	11	Dyspnea; hoarseness; cynaosis	5 wk.	?	Larynx not examined	Yes	History and angiomas elsewhere	Intubation; radium	Symptom free 2 mo. later	None
Levbarg.....	M	2½	Dyspnea; stridor; indrawing	2 days ?	Yes	Mouth and pharynx, involving larynx by extension	Yes	Laryngoscopy	Intubation; roentgen therapy	Dyspnea cleared; angioma present but smaller after several months	None

underlying cartilages. The mucosa of the larynx, trachea and bronchi was covered with a stringy, thick, sanguinopurulent exudate.

The lungs showed massive atelectasis, more marked on the left side.

The remainder of the gross necropsy findings were of no contributory importance.

Microscopic Observations: Sections of the laryngeal tumor mass showed the inner surface lined with a normal-appearing, noninterrupted laryngeal mucous

Photomicrograph (× 100) illustrating immature and mature capillaries, scattered submucosal glands and areas of poor differentiation.

membrane. Immediately subjacent to this was a narrow strip of rather loose edematous connective tissue, moderately infiltrated with round and plasma cells and containing normal-appearing, engorged capillaries. This loose connective tissue penetrated, in peglike projections, into the deeper structures, separating the tumor mass into large irregular islands of tissue.

The tumor itself was composed chiefly of closely packed capillaries. Many of these were mature in appearance, but others were in early stages of development.

The latter had lumens increasing from about 8 microns in diameter and contained from one to several red blood cells. Their walls were formed of one layer, or frequently two layers, of elongated, sausage-shaped, vesicular nuclei, with meager, pink-staining cytoplasm. The scanty and primitive type of intervening connective tissue, making up the stroma, was directly continuous with these capillary cells. There too the nuclei were vesicular, and round or elongated. The cytoplasm was also meager. In several areas the tumor cells had no alveolar or capillary arrangement and existed as masses of closely packed cells. Their nuclei were of the same nature as those just described. Mitotic figures were not encountered.

Scattered in the tumor mass were isolated laryngeal mucous glands. Many of them appeared somewhat hypertrophied. Others were distorted, as though compressed by the tumor cells. Most of them possessed a central lumen in which there was some bluish-tinged secretion. The cells themselves were cuboidal, or even columnar, with round, vesicular, basilar nuclei. In many instances the cytoplasm contained droplets of secretion.

Microscopic examination of a section of the brain through the pons and the fourth ventricle by Dr. Bernard J. Alpers revealed toxic cell changes in the abducens nuclei.

Comment.—We have designated the tumor in this case as a cellular hemangioma, yet in several respects it appears that it was more than this. In many areas the capillaries were mature, while in others they were primitive. The walls were often two layers in thickness. The lumens were small and either were empty or contained one or more red blood corpuscles. In several areas there was no sign of capillary formation. The stroma was not abundant, and in many portions a direct gradation could be traced from it to the endothelial lining of the capillaries. A diagnosis of capillary hemangioendothelioma could thus be well substantiated. Lynch [9] reported 7 cases of hemangioendothelioma of the larynx. His microscopic description is extremely brief, but the photomicrographs presented appear similar to those of our histologic sections. We maintain our diagnosis of a simple cellular hemangioma chiefly to conform with the diagnosis in the majority of cases reported.

COMMENT

Cause.—The neoplasms in cases of infantile hemangioma of the larynx are apparently congenital. Although chronic inflammation and vocal abuse followed by hyperplasia may be predisposing or etiologic factors of angioma in adults, this obviously cannot apply to infants. In Hofmann's [8] 2 cases the symptoms were present at birth, and in Sweetser's [4] case the first attack of dyspnea occurred at the age 9 days. The onset of symptoms shortly after recovery from measles in the case of Phillips and Ruh [5] was probably coincidental.

Sex Incidence.—The incidence with regard to sex appears to follow closely that found among adults. In this group 5 of the patients were male and only 1 female.

Symptoms.—The most prominent symptom is apparently obstructive dyspnea. In all cases in this series that was the case, the dyspnea being accompanied by inspiratory stridor, hoarse cry, croupy cough and vary-

9. Lynch, R. C.: Endothelioma of the Larynx, Tr. Am. Laryng. A. **51**:52, 1929.

ing degrees of laryngeal obstruction. There is little or no fever unless pulmonary complications arise. There may be either expectoration of blood-tinged mucus or gross external hemorrhage, although neither occurred in any of these cases prior to operative intervention.

It is interesting to note that in 5 of the 6 reported cases there was a history of one period or more of remission of symptoms, then an attack of rather severe obstructive dyspnea, followed by a period of well-being, only to be succeeded by another attack of dyspnea, usually more severe than the first. These relatively sudden changes in the clinical picture are probably dependent on changes in the volume of blood within the tumor from time to time. This was borne out in Hofmann's [8] second case, in that at the time of admission a roentgenogram revealed definite narrowing of the tracheal lumen from the left paratracheally, whereas three days later, before therapy had been instituted, a second roentgen examination failed to show the narrowing, and the infant had improved clinically.

Diagnosis.—The diagnosis in 4 of the 6 cases of proved hemangioma was established at autopsy. Study of the cases recorded in the literature indicates that a history of recurring attacks of obstructive dyspnea accompanied by little fever or other general systemic reaction and the laryngoscopic finding of a sessile, grayish red or bluish subglottic tumor in an infant should lead one to bear in mind the possibility of the presence of a hemangioma. The concomitant finding of angiomas elsewhere on the body should strengthen one's suspicions. A positive diagnosis, of course, is dependent on microscopic examination. In view of the danger of hemorrhage and local reaction after removal of tissue for biopsy from an already small airway, it is probably safer to do a preliminary tracheotomy.

Treatment.—For the relief of obstructive dyspnea a low tracheotomy is the procedure of choice, even when pneumonia is present. This should not be delayed too long if the patient is seen early, as the danger of pulmonary complications increases with prolonged retention of secretions, and the patient's resistance is lowered by the fatigue incurred by a long struggle for air. If a diagnosis of hemangioma has been made or if that disease has been strongly suspected, one should not be misled into delaying tracheotomy because of a characteristic period of remission.

Intubation has fallen into disrepute because (1) it is often difficult to perform, (2) the tube is frequently coughed out (Levbarg's case) and (3) there may be serious traumatic external hemorrhage or hemorrhage into the tumor itself, with more encroachment on the airway, as occurred in Sweetser's [4] case. Furthermore, intubation does not permit one to aspirate accumulated secretions as readily as does tracheotomy.

For eradication of the hemangioma, New and Clark [6] obtained good results with radium therapy applied over the front of the neck, employing 50 mg. with 2 mm. lead screening for five hours. Only one application was required. They recommended placing radium directly against the tumor by suspension laryngoscopy as the procedure of choice.

Levbarg used roentgen therapy in the treatment of a 10 week old boy who had a hemangioma of the mouth and pharynx which involved the larynx by extension. He was able to remove the intubation tube

eight days after giving one roentgen treatment. The exact amount of irradiation given was not stated. Hofmann's [5] experience with roentgen therapy was not so satisfactory. He gave a total of 400 roentgens in three exposures at four day intervals and noted marked improvement for sixteen days, only to have a return of dyspnea requiring tracheotomy.

Surgical procedures are not well tolerated by infants, but if the hemangioma should not respond to irradiation one would be confronted with making a choice between removal by endoscopic methods, which have been employed with some success on adults, and removal by thyrofissure. Obviously the latter would be the less dangerous, because better exposure is obtained and hemorrhage can be controlled. All authors agree that any such procedure should be preceded by tracheotomy. Hofmann [8] removed a hemangioma by tracheolaryngofissure one month after tracheotomy on a 2 month old infant, operating with the region under local anesthesia; no serious hemorrhage occurred, but there was suppuration of the wound, and the patient died one month later of pneumonia.

Complications.—Several complications may occur in association with juvenile hemangioma of the larynx. Five of the infants in these recorded cases died either of asphyxia itself or of pulmonary complications following procedure instituted to prevent asphyxia. One of the patients who died of asphyxia had accompanying lobar pneumonia, and for this reason tracheotomy was not performed. In Sweetser's [4] case the patient was asphyxiated because of hemorrhage into the tumor itself after unsuccessful intubation. Two of the infants died of pneumonia after surgical intervention and our patient of massive atelectasis in spite of tracheotomy.

SUMMARY

A short summary of the reported cases of hemangioma of the larynx in infants is presented. To these is added 1 more case. Histologically the growth is a cellular capillary hemangioma.

ACUTE SUPPURATIVE MASTOIDITIS

Sulfanilamide Therapy, Agranulocytosis and Death

JOSEPH KASNETZ, M.D., BROOKLYN

The literature contains an abundance of writings on sulfanilamide. The physical and chemical properties and the dosages of the drug,[1] the clinical indications,[2] the toxic properties,[3] and the distribution and concentration in tissues of the body [4] are well described in detail. Physicians in every branch of medicine have subscribed to it enthusiastically and have announced with great zeal and earnestness successful results of its use. Little has been said of failures with it, and no more than a dozen deaths have been reported.[5]

The otolaryngologist as yet has reported no fatalities. It is my sad lot to report the first death in a case of acute suppurative mastoiditis treated by sulfanilamide. The patient died from agranulocytosis which developed six days after cessation of the administration of the drug.

REPORT OF A CASE

A woman aged 45 years was admitted to the hospital on April 18, 1939. The history was of continuous otalgia on the right side for eleven days, otorrhea for

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From the Department of Otolaryngology of the Jewish Hospital, service of Dr. P. Leibowitz.

1. Molitor, H., and Robinson, H.: Pharmacologic and Toxicologic Properties of Sulfanilamide and Benzylsulfanilamide, J. Pharmacol. & Exper. Therap. **65**:405 (April) 1939.

2. (*a*) Long, P. H., and Bliss, E. A.: The Clinical Use of Sulfanilamide and Its Derivatives in the Treatment of Infectious Diseases, Ann. Int. Med. **11**:575 (Oct.) 1937. (*b*) Schenck, H. P.: Use of Sulfanilamide in Otolaryngology, Arch. Otolaryng. **28**:698 (Nov.) 1938. (*c*) Tunnicliff, R.: Action of Prontosil-Soluble and Sulfanilamide on Phagocytic Activity of Leukocytes and on Dissociation of Streptococci, J. Infect. Dis. **64**:59 (Jan.) 1939.

3. Schlesinger, E. R., and Mitchell, W. L., Jr.: Sulfanilamide Eruption: Study of Patients with Morbiliform Rash and of Their Subsequent Reactions, Am. J. Dis. Child. **56**:1256 (Dec.) 1938.

4. (*a*) Engelfried, J. J.: Further Observations on Distribution and Concentration of Sulfanilamide in Tissues of the Body After Enteral and Parenteral Administration, J. Michigan M. Soc. **38**:129 (Feb.) 1939. (*b*) Marshall, E. K., Jr.; Emerson, K., Jr., and Cutting, W. C.: Para-Aminobenzenesulfonamide: Absorption and Excretion; Method of Determination in Urine and Blood, J. A. M. A. **108**:953 (March 20) 1937.

5. (*a*) Plumer, H. E.: Severe Neutropenia Occurring During the Use of Prontylin, New England J. Med. **216**:711 (April 22) 1937. (*b*) Model, A.: Agranulocytosis and Para-Aminobenzene Sulphonamide, Brit. M. J. **2**:295 (Aug. 7) 1937. (*c*) Young, C. J.: Agranulocytosis and Para-Amino-Benzene Sulphonamide, ibid. **2**:105 (July 17) 1937.

nine days and severe headaches for the past two days. Myringotomies had to be performed twice, the second time because of inadequate drainage. For forty-eight hours before her admission the temperature varied from 101 to 103 F., and the pain behind the ear became excruciating. The patient complained also of periodic chills.

Examination on Admission.—The objective symptoms were those of an acutely ill patient. She appeared flushed and lethargic and was slow of speech. When the purulent exudate was mopped from the canal, the tympanum was found to be red and edematous, with a pulsating discharge. The nose, throat, heart, lungs, abdomen and extremities were normal.

Treatment and Course.—Sulfanilamide therapy (dosage and amounts to be discussed later in this paper) was begun, and the patient was observed for forty-eight hours. The temperature remained elevated, and the pain persisted. An operation was decided on, and a simple mastoidectomy was performed on April 20, 1939.

The mastoid process was found to be well developed and pneumosclerotic, and the pneumatization extended into the squama and the posterior zygomatic root. Although the cell spaces were filled with thick creamy pus, the intercellular walls were firm and resistant to the curet. Because of the unusually high temperature (103.6 F.) an inspection of the lateral sinus was made. The wall, which was exposed from the superior petrosal sinus down to the jugular bulb, was found to be deeply injected and showed evidence of inflammatory reaction. There were no granulations or pus. The dura of the middle fossa was also exposed in the region of the antrum and appeared normal.

For four days after the mastoidectomy no improvement was noticed. The temperature remained elevated (103 F.), and the pulse was rapid; the patient did not appear at all well. Aside from a slight nuchal rigidity, results of neurologic examination were negative. The mastoid cavity was infected, and free pus was draining from the wound and the auditory canal.

On the fifth day postoperatively it was decided to reinspect the mastoidectomy cavity and also to examine the spinal fluid. The mastoidectomy cavity was reopened and more of the sinus and dura uncapped. No additional pathologic condition was found. The spinal fluid was found to be clear, under slightly increased pressure and with a cell count of 40 lymphocytes.

After this procedure the temperature dropped to within the normal range and remained so for the next ten days. After four days of normal temperature, the administration of sulfanilamide was discontinued (April 29). Because of the extreme weakness and slight secondary anemia a transfusion of 300 cc. of whole blood was given by the direct method. After the transfusion the patient made steady progress. The mastoid cavity remained clean and was granulating in steadily. The auditory canal was now dry, and the middle ear showed steady resolution. There were no complaints of headache or pain; again the patient was neurologically normal. On May 4 I requested the resident physician to dismiss the patient to the outpatient department for further dressings.

The next day, when about to discharge the patient the resident physician found that she took a sudden turn for the worse. An erythematous rash appeared over the upper and lower extremities. There were numerous herpetic eruptions over the upper lip, and the temperature jumped to 105 F. Rhinopharyngoscopic examination showed a deeply injected mucosa with suggestive ulcerations of the tonsillar fauces. Medical and neurologic consultations resulted in no organic findings. A complete study of the blood was done, and a diagnosis of agranulocytosis was made.

Another transfusion of 300 cc. of whole blood was given. Three days later, May 8, pulmonary edema associated with cardiac collapse developed, and the patient died that evening.

Sulfanilamide Therapy.—A total of 80 Gm. of sulfanilamide was given from April 4, the day of onset of the illness, until May 8, the day of death. The dosage was: 40 grains (2.59 Gm.) per day for the first fourteen days, 80 grains (5.18 Gm.) per day for the next six days and 30 grains (1.94 Gm.) per day for the next four days, the last named period being that during which the temperature remained around 100 F. From April 29 on the use of the drug was totally discontinued. At no time during the entire period of injection of the drug did the patient show any sign or symptom of idiosyncrasy. Blood counts were performed at frequent intervals. No determinations of the blood concentrations were done.

Laboratory Data.—On April 18 the urine was acid, with a specific gravity of 1.012 and with no albumin, blood or pus cells. On the same date both the Wassermann reaction of the blood and the results of chemical analysis of the blood were normal. Culture of pus from the ear showed Staphylococcus aureus, and that of pus removed from the mastoid by the curet yielded Streptococcus haemolyticus. Culture of the spinal fluid was sterile, and no organisms were revealed by smear. Culture of the blood on two occasions, April 23 and 27, was sterile. On may 6 culture of the blood on broth revealed Staph. albus and on plate two colonies of Staph. albus, but the findings were probably due to contamination. Roentgenographic examination of the lungs, May 8, showed no evidence of a pathologic pneumonic process.

Results of the examinations of the blood are shown in the following table:

Date	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells	Polymorphonuclears, per Cent	Lymphocytes	Bands	Mononuclears
4/18	81	3,700,000	12,400	76	24	0	0
4/20	62	3,400,000	17,400	79	21	0	0
4/24	62	3,200,000	17,050	81	12	5	2
		After discontinuance of Sulfanilamide 4/29					
5/ 1	62	3,100,000	8,150	73	18	6	3
5/ 4	56	2,900,000	6,050	67	28	2	3
5/ 8	56	2,890,000	1,000	0	100	0	0

COMMENT

Apparently at present there are no forerunning warning symptoms of sulfanilamide poisoning. Therefore there are no means of ascertaining in which patient it may, and in which it may not, have fatal results.

The fatality does not depend on the size of the dose.

Administration of sulfanilamide seems to have the effect of producing a temporarily symptom-free period, followed by a sudden return, without warning, of all the severest symptoms.

Since it is well established that the diffusibility of sulfanilamide into osseous tissue is not so great as into soft tissue, the efficacy of the drug in treatment of acute infections of the middle ear and mastoid is still a question and the results of its use far from certain.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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ANATOMY AND PHYSIOLOGY OF THE EAR

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In reviewing the literature from October 1938 through December 1939 pertaining to the anatomy and physiology of the ear, we present summaries and abstracts of articles which permit the reader in a brief time to obtain a panorama of the field concerned. This review is not intended to be self sufficient, as are the original papers. Only articles of special value to us are included in this review.

DEVELOPMENTAL ANATOMY

The origin and distribution of air-containing cells in the temporal bone were investigated by Bast and Forester.[1] They approached the problem in a logical way, not only employing serial microscopic sections of temporal bones of adults but starting with those of the embryo and continuing with those of children up to 6 years of age. Ninety-seven bones of 69 fetuses and 39 bones of 27 infants were used.

The eustachian tube in the embryo extends to the middle ear by the eighth week, and then its epithelium invades slowly the connective tissue of the middle ear until it is completed late in fetal life. The antrum first appears in the twenty-second week of fetal life and is well developed by the thirty-fifth week. There is invasion of the tympanic plate in isolated areas that later fuse to enlarge the antrum. No true apical air cells were seen other than those of the postcarotid group, which extend forward into the apical part of the petrous bone. Occasionally mastoid cells open directly into the middle ear. The carotid group of cells open into the middle ear, while some of the subtubal and precarotid cells opened into the eustachian tube. There is also intercommunication between groups of air cells, especially between subgroups of carotid air cells.

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1. Bast, T. H., and Forester, H. B.: Origin and Distribution of Air Cells in the Temporal Bone, Arch. Otolaryng. **30**:183-205 (Aug.) 1939.

Air cells do not extend into true bone marrow. Formation of air cells occurs as a direct extension of epithelial buds into newly forming bone, or the process may invade older bone with bone marrow. In the latter case, bone marrow cells disappear, and loose, acellular connective tissue forms prior to the invasion of the epithelium.

Large spaces resembling air cells are sometimes seen in histologic sections of bone marrow, but they are not true air cells. They are due either to some pathologic change or to postmortem formation of gas.

By the Born wax plate method of reconstruction Wilson [2] has made an anatomic study of the air cells of the petrous portion of the temporal bone of a 13 year old child. This work continues his valuable studies on pneumatization. In this case the relationship to the middle ear is important. He found the air cells of the petrous bone to be well developed. They originated from two independent sources: (1) extension of the mastoid cells and (2) tubal cells from the hypotympanic space. These two groups of cells do not communicate with each other. The tubal cells were located in the inferior part of the petrous portion of the temporal bone in relationship to the carotid canal. They opened into the hypotympanic space by a distinct channel, or opening. According to Wilson, well developed tubal cells are correlated with well developed mastoid cells. It is agreed that the general pattern of pneumatization is practically complete in the mastoid area at the age of 5; thereafter there is only a slow increase in size. As yet there is not enough evidence to show when pneumatization of the petrous bone is complete.

Lettenbauer [3] examined by the roentgenologic technic of Stanvers and Meyer the temporal bones of 50 cadavers. Then sections were made of the petrous bones parallel to the posterior surface. He found marked asymmetry of the cells of the petrous tip on the two sides. Pneumatization extended to the tip in 15 pairs, but only 1 specimen showed complete symmetry. The same relationship held true for perilabyrinthine pneumatization. Peritubular cells were found in 14 specimens and were present in only 2 specimens without pneumatization of the tip. Whenever there was marked peritubular, perilabyrinthine and tip pneumatization there was also a very cellular mastoid process, but in the latter there was usually symmetry between the two sides. In men pneumatization was 50 per cent greater than in women. Pneumatization was found mainly in long heads and in those of medium length. The facial index is no morphologic guide. Heavy and com-

2. Wilson, J. G.: The Air Cells in the Petrous Temporal Bone in a Child of Thirteen Years, Ann. Otol., Rhin. & Laryng. **48**:35-38 (March) 1939.

3. Lettenbauer, W.: Pneumatization of the Petrous Bone: An Anatomical and X-Ray Study with Special Reference to Constitutional Factors, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:314-335, 1938.

pact skulls (commoner in women) are usually not well pneumatized. Comparison was made of the anatomic and roentgenologic appearances. The hope is expressed that further similar research relative to the living organism, with all its constitutional characteristics, will emphasize the importance of the arrangement of pneumatization from a clinical point of view. The value of serial roentgenograms in this connection is emphasized.

The morphologic and roentgenologic aspects of the temporal bone were studied by Jackson.[4] In his study of 536 bones he attempts to establish phases of development based on the amount of pneumatization. Standard roentgenograms were developed, from which nine measurements were taken. He established a working schedule arranged according to the amount of pneumatization and divided into the following phases:

1. Pneumatization commencing (birth).
2. Pneumatization moderate (two years).
3. Pneumatization extensive, not including apex (childhood, 6 to 14 years).
4. Pneumatization complete, including apex (adolescence).

Thus a standard based on the age of maturation is developed by a study of specimens selected from those which show the most extensive pneumatization for each stage. Investigation based on these specimens is not handicapped by any evidence of retardation in the process of pneumatization.

Jackson maintains that his study supported Wittmaack's theory of pneumatization, but he was unable to find any specimen in which the apex was pneumatized earlier than 16 years of age. Fourteen per cent of the bones of adults showed pneumatic apexes, and there was wide variance between the two mastoids in individual specimens. One might be fully developed while the other might have stopped maturing at infancy or early childhood because of infection. There was no specimen in which the apex was pneumatized in which there had been arrested development of the mastoid cells.

In studying 20 temporal bones before and after maceration, he found that one is unable to differentiate between marrow cells and pneumatic cells, save that the latter, being ballooned, have a different outline.

Anson and his associates [5] present wax reconstruction models, drawings and sectioned photomicrographs to demonstrate the stapes, fissula

4. Jackson, C. C. R.: Morphologic and Roentgenologic Aspects of the Temporal Bone, Arch. Otolaryng. **28**:561-580 (Oct.); 748-767 (Nov.) 1938.

5. Anson, B. J.; Karabin, J. E., and Martin, J.: Stapes, Fissula Ante Fenestram and Associated Structures in Man, Arch. Otolaryng. **28**:676-697 (Nov.) 1938; **29**:939-973 (June) 1939.

ante fenestram and associated structures in man. The study describes the development of the stapedial area in embryos and fetuses from 22.8 mm. length to 183 mm. length. In the 22.8 mm. embryo the stapedial cartilage is clearly defined and in the shape of a ring; while in the 183 mm. fetus it nears the adult size and shape. The window of the fissula reaches its definite shape and size in the 161 mm. fetus. The window is lined by a rim of cartilage throughout life, which decreases as age advances, but at no time is it entirely replaced by bone. The fissula ante fenestram and the fossula post fenestram are surrounded by cartilage which is directly continuous with the cartilaginous rim of the fissula. The fossula post fenestram occurred in but 1 of the 3 older specimens studied. Here it lay within a posterior projection of the fenestral cartilage, which was considerably smaller than the chondral covering of the fissula in the same specimen.

The stapes was found to have attained adult dimensions in the fetus of 21 weeks, and after that there may be considerable variation in its later stages. The proportion of cartilage to bone changes from 3 to 1 or 2 to 1, in the fetus at term, to 1 to 2 in adults. Throughout life changes are occurring. The base becomes thinner and riddled by invasive bone; in a like manner the overlapping cartilage of the lamina retreats from the basal ends of the crura, which become thinner as age advances. The neck and head of the stapes are still continuous parts of a solid cartilaginous cylinder in the 21 week fetus, at which stage the crura have become hollow columns composed entirely of bone. The hollowing of the neck of the head of the stapes is prominent and constant. In the submucosal tissue which surrounds the stapes, blood vessels are frequently abundant and are traceable into the corresponding layer on the tympanic aspect of the base, where they enter haversian spaces.

Further study has been conducted by Anson and Wilson,[6] in which they used their serial sections of temporal bones and wax plate reconstructions. They point out that the temporal bone as a whole undergoes progressive changes which continue throughout life. The changes consist in growth and internal alteration in the otic capsule and the associated stapes.

Centers of ossification in the otic capsule coalesce by midfetal life and grow little after that, while the petrous portion continues to enlarge after birth. In a similar way the vestibular window has attained adult size during the first half of fetal life, as has the stapes. The rim of the hyaline cartilage which lines the window is gradually reduced as age advances.

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6. Anson, B. J., and Wilson, J. G.: Structures of the Petrous Portion of the Temporal Bone, with Special Reference to the Tissues in the Fissular Region, Arch. Otolaryng. **30**:922-942 (Dec.) 1939.

In the area between the vestibular window and the cochlea is the fissula ante fenestram, where abnormal changes occur with such regularity that it is considered clinically to be the site of predilection for otosclerosis. This area is not larger than 1 cu. mm. Anson and Wilson state:

> The fissula ante fenestram is typically an irregular channel through the temporal bone situated in front of the vestibular window; in the cleaned skull it is an open channel, admitting a bristle, but in the natural state it is occupied by fibrous tissue. Its immediate wall is formed by cartilage, which like the cartilaginous content of the stapes or the chondral lining of the vestibular window, is a remnant of the originally larger collection of the same tissue, gradually encroached on by bone.
>
> But the changes are not merely those of slow replacement of one tissue by another; certain other, more rapid alterations occur, which mark the area as histologically unstable. These seem to be related fundamentally to the secondary formation of hyaline cartilage, derived from the cartilaginous lining of the fissular channel. Displacing the fibrous tissue of the fissula, the nodule of newly formed tissue extends through the length of the original channel; it may not only extend to the regular tympanic and vestibular orifices but also occupy the auxiliary opening at the vestibular window. Under these circumstances the cartilaginous lining of the fissula and the encapsulating bone are the site of only the slightest activity. Here an intermediate tissue is present, derived from hyaline cartilage but usurped by bone; this tissue, formed initially as cartilage, is invaded by osteoblasts, which convert the matrix into a calcified structure. Seemingly resistant to rapid change, this hybrid tissue ("intrachondrial bone" or "cartilage islands"), gradually incorporated into the wall of the fissula, remains readily distinguishable from the mass of activated cartilage, when such occurs; one is quiescent in character and constitutes part of the fissular wall; the other is active and is situated within the fissular channel; they seem independent, the less active being resistant to the more active tissue.
>
> But within the fissular channel another type of tissue occurs which is sufficiently invasive to replace, in succession, any fibrous tissue remaining in the fissular channel, the newly formed cartilage located there, the older hyaline cartilage and intrachondral bone which immediately borders the fissular wall. This mildly invasive tissue, much more vascular than the other tissues of the otic capsule, spreads beyond the normal confines of the fissula, occasionally to reach the anterior wall of the vestibular window and to reduce the latter's space. After having taken the place of the hyaline cartilage which forms the primordial fenestral wall, the sclerotic bone, should it impinge on the base of the stapes, would fasten the ossicle within the window.
>
> The total area affected by these processes is relatively small, so that an otosclerotic nodule of minute proportions may profoundly alter the equally small window and stapes; it is, so to speak, a pathologic process on a diminutive scale.

In infancy direct vascular connections between the submucosa of the middle ear and the dura are commonly found. With pneumatization there is an involution of these vascular connections, and they change to strands of connective tissue. If pneumatization is disturbed, or incom-

plete for other reasons, these vascular connections may persist. Steurer [7] studied persistent vascular connections, both gross and histologic. He found that persistent vascular connections often form a diagonal link from the region of the antrum up to the posterior part of the region of the sinus-dura angle; some vessels may open into the dura more anteriorly. The diameter of the vessel varies in thickness from the size of the finest of sewing thread to that of a knitting needle. The length ranges from 2 to 7 mm. The author states that clinical examination, roentgen examination and observations at operation revealed that these persisting vascular connections were mostly in patients with a dense mastoid containing few, if any, air cells. Periphlebitis or purulent thrombosis of these abnormal vessels leads to dural infections and meningitis. Dangerous persistent vascular connections should be watched for when disturbances of pneumatization are evident.

In an attempt to determine the pathways of infection between the internal ear and the meninges, Perlman and Lindsay [8] studied 58 temporal bones from 30 patients who had died of meningitis in the acute stages. They observed the extent of the collection of pus cells from the meninges toward the temporal bone and in this way demonstrated the pathways of infection in reverse. Usually one considers infection as going from the middle ear inward, and not from the meninges outward.

Their sections showed, first, a collection of pus cells in the subarachnoid space about the seventh and eighth cranial nerves in the internal auditory meatus, without destruction of the arachnoid. A subarachnoid space can usually be traced to the fallopian canal at the internal auditory meatus, sometimes as far as the geniculate ganglion, but rarely beyond. Just beyond the beginning of the foramen, the pia-arachnoid becomes closely applied to the trunk of the facial nerve to form its perineurium, thus ending the space. There is a similar distribution of the arachnoid about the eighth nerve, this obliterating the subdural and subarachnoid spaces.

Second, there is an invasion of the cochlear aqueduct and the appearance of pus cells about its opening into the scala tympani, without destruction of the arachnoid meshwork in the aqueduct. After this there is a diffuse invasion of cells along the nerves and vessels going through the lamina cribrosa and into the perilymphatic space. It is not known whether this is a diapedesis of cells through the membranes or a condition following a destruction of the pia at these points of ingress.

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7. Steurer, O.: Importance of Persisting Vascular Connections Between Cavities of the Middle Ear and Dura for Origin of Meningitis, Hals-, Nasen- u. Ohrenarzt (pt. 1) **30**:44-50 (Jan.) 1939.

8. Perlman, H. B., and Lindsay, J. R.: Relation of the Internal Ear Spaces to the Meninges, Arch. Otolaryng. **29**:12-23 (Jan.) 1939.

Later, cells are noted in both the scala tympani and the scala vestibuli of all coils, concentrated near the modiolus. Tumor cells and blood cells do not show this extension from the subarachnoid spaces into the modiolus.

The authors feel that the probable flow of cerebrospinal fluid is from the subarachnoid space into the scala tympani.

In another experimental work to determine the pathways of infection from the middle ear to the internal ear Ross and Hamilton [9] injected 8 per cent mercurochrome into the middle ear of anesthetized dogs. After a specified time had elapsed, the solution was fixed in the tissues with acid, and the bones were then decalcified and sectioned. It was observed that the intensity of staining was an indication of the path of invasion of the dye. It was found that the dye penetrated the entire cochlea. Tissues were stained in the following order: (1) bony wall of middle ear; (2) fenestra rotunda; (3) nerve cells of spiral ganglion, spiral lamina and eighth nerve; (4) stria vascularis; (5) articulation of the stapes; (6) meshes of the aquaeductus cochleae.

The lateral sinuses are the major venous channels of the brain. A knowledge of their variations is valuable to the otologist. The Tobey-Ayer test to determine venous sinus thrombosis is heavily relied on. Variations in the size of the lateral sinuses modify the result of this test. Woodhall [10] examined 100 brains to determine the anatomic variations in the venous pattern, especially in the region of the torcular Herophili and the lateral sinus.

The sinus patterns of the torcular Herophili and its contiguous sinuses fell into four general groups:

1. The common pool type. In this variation the longitudinal and straight sinuses meet in a more or less common pool, from which the blood is directed to the lateral sinuses. The latter are ordinarily equal but may vary in size within narrow limits. Nine instances of this simple type of conformation of the sinus were observed in this series, a number that closely agrees with that given in previous reports.

2. The plexiform type. The longitudinal and the straight sinus may each be divided into two parts, equal or unequal, and the lateral sinuses may be equal in size, but usually are moderately dissimilar. The straight sinus may be divided into two branches, equal or unequal, with the longitudinal sinus single, the latter flowing usually to the right with the smaller half of the straight sinus, or this arrangement may be reversed. The longitudinal sinus may be divided into two equal or unequal branches

9. Ross, E. L., and Hamilton, J. W.: Penetration of the Inner Ear by Mercurochrome, Arch. Otolaryng. **29**:428-436 (March) 1939.

10. Woodhall, B.: Anatomy of the Cranial Blood Sinuses with Particular Reference to the Lateral, Laryngoscope **49**:966-1010 (Oct.) 1939.

with the straight sinus single, the latter usually flowing to the left with the smaller branch of the longitudinal sinus. There is generally a more pronounced difference in the size of the lateral sinuses in these last two subtypes. There were 56 examples of this type of sinus pattern.

3. The ipsilateral type. The longitudinal sinus runs to one side, usually the right, and the straight sinus to the opposite side. Circulation between the lateral sinuses, if present, must take place through an additional direct channel or through the formation of a foramen, both structures varying considerably in size. In the majority of cases the lateral sinus on the side of the longitudinal sinus flow will be much the larger. This type of sinus pattern was noted in 31 preparations and was viewed with particular interest because of the character of its intersinus communications at the torcular Herophili. The longitudinal sinus entered the right lateral sinus undivided in 26 of these specimens and the left lateral sinus in the remainder. The straight sinus naturally followed the opposite course. The right lateral sinus was equal in volume to the left in 15 specimens, slightly larger in 6, twice as large in 5, three times as large in 1 and four times as large in 1. In 42 per cent of the specimens, then, there was a right-sided overdevelopment, frequently of a major character.

4. The unilateral type. In this extremely rare conformation both the longitudinal and the straight sinus empty into one lateral sinus. The opposite sinus may be attenuated, being fed only by minute venous channels in the adjacent dura, or it may be completely absent. There were found 4 examples of this rare conformation of the sinuses in the 100 dural preparations studied.

All of the foregoing variations and a few other rare abnormalities are well described, with diagrams to make the text more vivid. Woodhall points out that a roentgenogram of the marking of the occipital bone clearly shows the differences in the volume of the lateral sinuses and follows the exact distribution of these sinuses.

If roentgenograms of normal subjects are compared with the results of the Tobey-Ayer test there is a striking uniformity. The roentgenogram is a reliable index of sinus volume. In the relatively few cases in which sinus markings are not visualized in the occipital projection, a roentgenogram of the base of the skull will show the relative size of the jugular foramens, which is an equally valuable evidence of sinus volume.

Seventy cadavers were studied by Brunetti and Caramagna [11] to determine the origin of the internal auditory artery. In 70 per cent of the specimens they found that the internal auditory artery springs by a single trunk from the inferior cerebellar artery where it crosses the facial-

11. Brunetti, F., Jr., and Caramagna, V.: Origin and Behavior of the Internal Auditory Artery, Valsalva **14**:413-428 (Sept.) 1938.

acoustic trunk. Occasionally the internal auditory artery has two or three trunks. Very rarely does it spring from the basilar artery, as stated in older texts on anatomy. Stopford, in 1916 stated that the internal auditory artery comes from the inferior cerebellar artery.

The surgical treatment of otosclerosis consists of the creation of a fistula into the semicircular canal. Hearing is immediately improved. If the fistula remains patent this improvement in hearing may well be permanent. Nager [12] made histologic studies of the labyrinthine capsule of monkeys operated on by Holmgren. He demonstrates the method of healing these operative fistulas. The labyrinthine capsule of the monkey is very similar to that of man. Nager found in studying specimens obtained from twenty-one days to one year after the creation of the fistula that in all instances the fistula was closed with new-formed bone. This bone comes from the periosteum. Not only is the fistula closed, but the new-formed bone often grows into the lumen of the semicircular canal and obliterates it. There is no histologic evidence of reaction in the perichondral layer. No new bone was formed from the endosteum. Various substances used in the operation failed to keep the fistula open, and even radium emanation failed to inhibit (although it reduced) the formation of new bone.

Perlman [13] created perforating injuries to the labyrinthine capsule in rabbits and observed that they heal in a way resembling the healing of injuries to bony structures elsewhere in the body. An important observation was that these injuries did not create a progressive obliterative process within the labyrinth. His summary is:

> A small perforating injury of the capsule of the labyrinth is sealed by a blood clot after a few hours. Swelling of the adjacent periosteal layer is observed after the first day. The fibrous tissue proliferating from the periosteum then invades the blood clot and replaces it in three days. At the same time a differentiation of the periosteum into osteogenic layer, or cambium, and fibrous layer occurs. By the fifth day apposition of new bone begins against bone chips and the surfaces of the fracture as well as on cortex where the periosteum was injured. By the end of the second week most linear fractures are well filled with bone, along with vascular tissue, which has grown in with it from the periosteum. These blood vessels continue into the depth of the perforation, probably anastomosing with the intralabyrinthine vessels. The new bone and its nutrient vessels remain distinct from the adjacent bone of the capsule. At the end of four weeks excess periosteal callus and to a lesser degree endosteal callus are removed, and the activity of the tissues at the site of injury appears to have reached equilibrium. New bone gradually invades and replaces them without definite evidence of osteoclastic absorption in it or in the periosteal callus. Few fragments or spicules

12. Nager, F. R.: Demonstration of Holmgren's Operation for Otosclerosis on Monkeys, Acta oto-laryng. **27**:350-358, 1939.

13. Perlman, H. B.: Process of Healing in Injuries to Capsule of Labyrinth, Arch. Otolaryng. **29**:287-305 (Feb.) 1939.

are to be seen in the callus after thirty days. The endochondral and the endosteal layer of the capsule forming the walls of the perforation show no evidence of osteogenic activity. The connective tissue of the endosteum and the connective tissue covering the lamina between the coils and in the spiral ligament respond to injury by laying down a meshwork of delicate fibrous tissue, in part of which ossification may begin by the eighth day. Some of the spiral ligament itself at the site of the injury and for some distance away ossifies. Small localized perforating aseptic injuries to the cochlea of the rabbit do not result in a progressive obliterative process within the labyrinth, such as is produced by a suppurative process within the labyrinthine spaces.

Canfield,[14] however, claims that the response of vital bone to a defect depends on the way in which the defect was made and not on the type of tissue that is placed over the defect; that is, a defect made with a sharp instrument has a tendency to bony regeneration, whereas that made with the dental polishing burr does not. Probably in the latter case regeneration is only slowed down. Further lapse of time in his experiments will prove this point.

The otic capsule consists of three bony layers. The knowledge of the enchondral layer is summed up by Nager [15] as follows:

1. With the exception of the small ossicles of the middle ear this type of bone occurs in no other locality of the adult skeleton.

2. It is to be regarded as an arrested stage in the normal development of bone in cartilage.

3. It is completely formed in the newborn, and its appearance is not materially changed throughout life.

4. It is poorly vascularized bone and its metabolism must be of an extremely low grade.

5. It does not participate in the usual processes of resorption, deposition and healing of bone.

In order to study further the nature of the layer of the otic capsule Covell [16] carried out the following experimental procedure. Small fragments of bone from the region of the oval window margin of one rabbit were transferred to the anterior chambers of the eyes of other rabbits, and changes were observed in the transplants over a period of seven months. In this way it has been possible to ascertain the effects of depriving the bone of its blood supply, the changes in the bone when the vascularity becomes reestablished and the ability of the transplant to

14. Canfield, N.: Labyrinthine Fistulas, Arch. Otolaryng. **30**:50-62 (July) 1939.

15. Nager, F. R.: Pathology of the Labyrinthine Capsule and Its Clinical Significance, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1939.

16. Covell, W. P.: Experimental Studies on Enchondral Bone of the Otic Capsule, Ann. Otol., Rhin. & Laryng. **48**:934-939 (Dec.) 1939.

form new bone. He concluded that the endochondral bone of the otic capsule is incapable of forming new bone. In young transplants it undergoes necrosis, while in older transplants there is some tendency for it to return to its usual appearance.

Mellanby,[17] in a series of publications since 1926, has shown that degenerative changes in many nerves of both central and peripheral systems of young animals can be readily produced by dietetic means. The most common lesions consist of demyelination changes followed by complete disappearance of the fibers in many of the afferent peripheral nerves and the ascending fibers in the central nervous system. It has been shown that the nerve cells are also affected by dietetic conditions, and reasons have been given which suggest that the changes in the nerve cells may be primary to the demyelination of the nerve fibers.

In the usual experimental period of about four months, the afferent fibers degenerate before the efferent, which may not be much affected, if at all. With a longer dietary period the efferent fibers are affected, but never as badly as the afferent.

Although the most obvious lesions consist of changes in the myelin sheath, there are also changes in the axis-cylinders in the nerves of both dogs and rabbits fed on diets deficient in carotene and vitamin A.

One of the afferent nerves affected by demyelination changes under these dietetic conditions is the eighth, both the cochlear and the vestibular division; the former is affected more than the latter. Degenerate changes in the vestibular nerves are associated with incoordinate movements of locomotion of the animal, as might be expected. Young dogs showing this condition are also inattentive and do not respond to the call of people at close range, a characteristic which distinguishes them from normal puppies. This inattention is doubtless due partly to deafness.

The paper is concerned with these abnormal changes due to deficiency of vitamin A and contains a description of the pathologic condition of the labyrinthine capsule, both of the nerve supply and of the bone itself. When a rich source of vitamin A is added to the diet while every other condition of diet and environment is kept constant, the animals remain normal in behavior and the labyrinthine capsule escapes pathologic change.

Mellanby's summary is:

> The histological examination of fifty-one labyrinthine capsules of young dogs fed for some months on diets of natural foodstuffs but deficient in vitamin A and rich in cereals revealed the following changes:
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> a. Degeneration of different degrees up to complete disappearance of the cochlear nerve, the cells of the spiral ganglion and their central and peripheral branches.

17. Mellanby, E.: The Experimental Production of Deafness in Young Animals by Diet, Laryngoscope **49**:1090-1117 (Nov.) 1939.

b. Degeneration, but to a lesser degree, of the vestibular division of the eighth nerve.

c. Overgrowth of bone in the modiolus and of the periosteal layer of the capsule near the brain.

This overgrowth of bone is apparently responsible for the degenerative changes in the nerves by reason of the pressing and stretching of these tissues.

Serous labyrinthitis also develops in the cochlea of the dogs on a vitamin A deficient diet. This condition seems to produce degeneration of the sensory epithelium of the labyrinth, including that of the organ of Corti and of the ampullae of the semicircular canals, in course of time. The bone and nerve changes precede the development of the labyrinthitis.

Substitution of potato for the cereal element of these vitamin A deficient diets greatly reduces the abnormal changes in the labyrinth above described.

Examination of the base of the skull of these vitamin A deficient dogs reveals other bone overgrowth and deformity, which is probably responsible for the degenerative changes of other cranial nerves, such as the optic and trigeminal nerves, previously described.

Selfridge [18] has continued his work on the relationship of the eighth nerve to nutritional deficiencies. In a study of 30 patients for whom nicotinic acid, nicotinamide or sodium nicotinate was used in treatment he found a striking improvement in hearing. In all cases in which the audiogram shows a gradual decline in the tone scale, one is justified, the author states, in using these fractions, because, while improvement in the audiometric curve may not be very marked, the physical and mental improvement is sufficient justification. Nicotinic acid acts as a vasodilator, not only influencing the eighth nerve but also playing a part in removing the calcium from the adhesions around the footplate of the stapes in cases of chronic progressive deafness and perhaps also in cases of beginning heavy fixation of the stapes.

Selfridge found thiamine hydrochloride an essential addition to nicotinic acid. The influence of the other components of the vitamin B complex has not as yet been worked out. He found that in every case of eighth nerve deafness there was a deficiency of the output of vitamin B_1. All of these persons gave a history of faulty diet. He emphasizes that the obvious dysfunctions of the endocrine glands should be cared for, as also other vitamin deficiencies, abnormal level of electrolytes and any lowering of the intake of protein. He feels strongly that nutritional deficiencies offer the best explanation for the cause of the degenerative process involving the eighth nerve. In other words, Dr. Selfridge advo-

18. Selfridge, G.: Nicotinic Acid and the Eighth Nerve: A Preliminary Report, Ann. Otol., Rhin. & Laryng. **48**:39-53 (March) 1939; The Eighth Nerve in Relation to Thiamin Chloride and Nicotinic Acid: A Comparative Study, ibid. **48**:419-432 (June) 1939; Eighth Nerve High Tone Deafness from a Nutritional Standpoint, ibid. **48**:608-631 (Sept.) 1939.

cates that the general physical condition of the patient plays an important part in the amount of deafness from which he is suffering.

An eminent authority, Marburg,[19] has written a paper entitled "Modern Views Regarding the Anatomy and Physiology of the Vestibular Tracts." This article is on the "must" list for any one interested in neuro-otology.

PHYSIOLOGY OF HEARING

An excellent discussion of the physiology of hearing based on the histology of the structures involved is presented by Lurie.[20] He has correlated the results of his many years of experimental work at the Harvard Medical School, together with the work of other distinguished investigators in this field. His paper is limited to a discussion of perception deafness. By perception or nerve deafness is meant the inability of the end organ or of the brain to receive and interpret the sound waves. The evidence points in this type of deafness, not to the brain's inability to interpret sounds, but rather to a defect in the perception and the transformation of sounds into accurate nerve impulses. A study of the neuroepithelium of the perception organ (organ of Corti) does much to explain this type of deafness. Among the many facts about the organ of Corti that Lurie emphasizes, these stand out:

The tectorial membrane is really a modified otolithic membrane. In function it aids in the movement of bending of the hairs in the external and internal hair cells. The organ of Corti rests only partially on the basilar membrane. The external hair cells and the external pillar cell rest on the membrane; the internal hair cells and the internal pillar cell rest on bone. Thus greater movement is permitted the external hair cells and external pillar cell than the internal hair cells and pillar cell. Experiments which cause the basilar membrane to be vibrated for long periods create changes in the external hair cells before any changes are evident in the internal hair cells. Apparently a very small movement of the basilar membrane excites the external hair cells. The inner hair cells resting on bone require greater distortion of the organ of Corti. When by such experiments (mechanical stimulation) a degeneration of the external hair cells is obtained, the animals are not totally deaf, but lose only part of their hearing. This is attributed to the fact that the internal hair cells still respond to sounds of higher intensity. Thus the external hair cells are shown to respond to stimuli of a weak nature. The internal hair cells function when intensity of sound is raised 30 to 40 decibels above the threshold.

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19. Marburg, O.: Modern Views Regarding the Anatomy and Physiology of the Vestibular Tracts, Laryngoscope **49**:631-652 (Aug.) 1939.

20. Lurie, M. H.: What Is Perception Deafness from a Physiological and Histological Basis? Ann. Oto., Rhin. & Laryng. **48**:3-16 (March) 1939.

Animals made deaf by the use of quinine also showed similar degeneration of the external hair cells. This can be taken to indicate that the external hair cells are more likely to degenerate from toxic poisoning by drugs or disease than are the internal hair cells.

Then if one considers perception deafness to be a degeneration of the external hair cells in the organ of Corti, a scattered degeneration will cause slight loss of hearing and a more widespread degeneration a greater loss.

Complete loss of hearing results when there is a degeneration of both the external and the internal hair cell. Histologically the inner hair cell is larger than the slender, longer external hair cell. These two special types of hair cell have different nerve supplies. A single nerve innervates not more than one or two hair cells; in contrast, a nerve to the external hair cell supplies a larger number (Lorente de Nó [21]) of cells. This means that sounds of small intensity affecting the basilar membrane will be picked up by the external hair cells with a multiple nerve supply. With a multiple nerve supply, slight changes are easily detected, but no fine discrimination of pitch is present. The single nerve supply of the internal hair cells indicates accurate determination of pitch, for a difference in pitch can be detected two or three hair cells apart.

Physiologic tests show that this determination of pitch at intervals below 30 decibels is about 500, compared with 1,500 different pitches appreciated at levels of intensity above 60 decibels.

When the nerve itself or the ganglion of the cochlear nerve degenerates, deafness, of course, results. This type of deafness is of the complete type. It is interesting to know that the organ of Corti can be shown to function for long periods (three months) after the cochlear nerve has been damaged. The ability of the organ of Corti to survive damage to the nerves may explain the recovery of hearing in patients who have had complete deafness during the course of a toxic disease.

Lurie's [22] work with waltzing guinea pigs has shown that their circling and deafness are inherited characteristics of the recessive type. This may mean a revision of the conception of human deafness, in that it may be due to an inherited degenerative factor. Thus one has an explanation for inherited deafness beginning in childhood, in puberty or later in life which from its inception does not give the picture of true otosclerosis. It is of interest that the vestibular mechanism in

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21. Lorente de Nó, R.: Anatomy of the Eighth Nerve, Laryngoscope **43**:1-38 (Jan.) 1933.

22. Lurie, M. H.: Studies of the Waltzing Guinea Pig, Laryngoscope **49**:559-565 (July) 1939.

the waltzing guinea pig is normal and that the animal's circling movement is caused not by vestibular involvement, but by a disarrangement of the central nervous mechanism, probably in the region of the red nucleus. This is borne out by experimental work (Lurie and Dempsey [23]).

Further, with waltzing guinea pigs there is no evidence that the saccule is concerned with sound. The organ of Corti degenerates, and, as stated, this is an inherited characteristic. Experimentally the author has shown that the cochlear nerve can degenerate (by cutting) without degeneration of the organ of Corti. In guinea pigs with complete absence of the organ of Corti, ganglion cells and nerve are shown to be present. This indicates that the organ of Corti is not bound up as intimately with its nerve supply as has been thought. In the guinea pig the degeneration starts in the organ of Corti at or near birth and then spreads to involve the cochlear nerve.

In cases of paralysis of the facial nerve, occasional patients have increased sensitivity to sound. Neurologists have long felt that this was due to paralysis of the stapedius muscle. Visible proof has been lacking until recently. The contraction of the stapedius muscle in the human in response to acoustic stimuli was first observed in 1929, by Luescher.[24] He found that a visible contraction of the stapedius muscle did not occur at low intensity levels of sound, but that a definite above threshold level was necessary. In 1936 Lindsay, Kobrak and Perlman [25] published the first quantitative studies of the sound intensities needed to obtain a just visible contraction of the stapedius muscle. They found that the threshold of the muscle contraction was about 65 decibels above the threshold of hearing. As a result, it became clear that the stapedius muscle very likely did not enter into the mechanism of hearing sounds under 65 decibels above threshold, in intensity, but that its function was probably that of protection of the cochlea when sounds of sufficient intensity and of sudden onset reached the ear.

In the light of this knowledge of the function of the stapedius muscle Dr. Perlman [26] reports the quantitative effect of paralysis of the stapedius muscle in a patient who had unilateral, uncomplicated facial paralysis. This patient complained of increased sensitivity, or hyperacusis. He

23. Lurie, M. H., and Dempsey, E. W.: Experimentally Induced Circling (Waltzing) in the Guinea Pig, Laryngoscope **49**:565-571 (July) 1939.

24. Luescher, E.: Die Funktion des Musculus stapedium beim Menschen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:105, 1929.

25. Lindsay, J. R.; Kobrak, H., and Perlman, H. B.: Relation of the Stapedius Reflex to Hearing Sensation in Man, Arch. Otolaryng. **23**:671-687 (June) 1936.

26. Perlman, H. B.: Hyperacusis, Ann. Otol., Rhin. & Laryng. **47**:947-953 (Dec.) 1938.

was tested as to hearing sensation for threshold and over threshold sounds, auditory fatigue, masking and "adaptation."

The onset of hyperacusis occurred constantly at a definite intensity level for various frequencies, an intensity level which corresponds with the normal threshold for contractions of the stapedius muscle. The sounds which the patient described as causing discomfort were of an intensity and sharpness to produce contractions of the stapedius muscle. Fatigue of the auditory mechanism was more marked in the affected ear, which required a longer period for recovery.

The phenomenon which might be described as "adaptation" could be demonstrated; namely, prolongation of an intense sound was accompanied by disappearance of the "discomfort" in a period closely corresponding to that in which the stapedius contraction is known (experimentally) to disappear, thus bringing about an equality of loudness in both ears. The study of these phenomena as afforded by this clinical case corroborates the findings previously reported on the action of the stapedius muscle and clarifies the conception of the part played by the stapedius muscle in the function of hearing.

The author states that there are two types of hyperacusis: first, a definite lowering of the threshold of hearing, or increased acuity which may be designated oxyecoia; second, an abnormal discomfort caused by sounds above certain intensity, for which the term hyperesthesia dolorosa has been suggested. The latter type is now proved to be due to paralysis of the stapedius muscle.

It is now established that the reflex contractions of the stapedius muscle to sudden loud sound stimuli protect the cochlear structures from damage by diminishing the amplitude of vibration of the stapes. This reflex contraction is initiated in response to a certain number of nervous impulses arising in the cochlea and transmitted along the reflex pathway. During the time needed for these reflexes to complete the reflex arc, from the onset of sound to the onset of muscle contraction, the cochlear mechanism is completely unprotected. Perlman and Case [27] have measured this latent period for the first time in man by recording the action potentials of the contracting stapedius muscle. The latency of the human stapedius muscle reflex for a loud tone of 1000 cycles is about ten and a half milliseconds. Perlman and Case direct attention to the importance of this latent period in explaining the increased degree of acoustic trauma developing in the presence of rapidly repeated loud sounds, as in the experimental animal and as in operators of pneumatic hammers, metal stamping machines, rapid fire arms and other similar instruments.

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27. Perlman, H. B., and Case, T. J.: Latent Period of the Crossed Stapedius Reflex in Man, Ann. Otol., Rhin. & Laryng. **48**:662-675 (Sept.) 1939.

Pohlman [28] insists that the mechanics of middle ear structures in transmitting sound is still not understood. He feels that the theories of the physiology of hearing laid down by Weber and by Helmholtz are not tenable. Pohlman maintains that we must start anew in the formulation of ideas regarding the function of auditory structures.

Dickson and his associates [29] have made audiometric determinations of the effect of noise produced by airplanes on hearing. They found that persons exposed to the noise of airplane engines suffered a loss of hearing for high tones if their ears were not protected. This loss of hearing varies with the individual. It becomes apparent after a few hundred hours, is at first temporary but after a time remains permanent. The deafness does not amount to disability until the speech frequencies become involved. Bone conduction is diminished over the frequencies that show a loss by air conduction. The authors discovered an apparent inconsistency with evidence obtained in experiments on aural fatigue and cochlear degeneration as the result of noise.

Experimental work has shown that depression of auditory response occurs at, or about, the frequency of the fatiguing tone. These authors found that wave analysis of aircraft noise indicates that the components are of large amplitude low in frequency. If this is true it would mean that a low-pitched noise (airplane noise) is causing high tone deafness. Elucidation of this apparent inconsistency may be of importance in our understanding of cochlear function. If a high-pitched component is later found present in airplane noise, animal experiments will be confirmed.

It is reassuring that protection of the ears diminishes and probably eliminates the risk of impairment of the auditory acuity of occupants of aircraft. A high altitude type of flying helmet firmly strapped so that the telephones and their holders closely seal the ears protects against high tone loss. If this type of helmet is not used, packing the meatus with cotton smeared with petrolatum is a quick, safe and efficient method of protection.

Several authors have published their findings dealing with the immediate response to the sound of a pistol shot. The reflex reactions were, on the whole, of the same pattern. Their observations varied, as they depended on the unaided eye to detect very quick motions. Strauss and his associates,[30] dealing with psychiatric patients and using

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28. Pohlman, A. G.: Restoring Forces in the Mechanics of Audition, Ann. Otol., Rhin. & Laryng. **48**:148-157 (March) 1939.

29. Dickson, E. D. D.; Ewing, A. W. G., and Littler, T. S.: The Effects of Airplane Noise on the Hearing Acuity of Aviators: Some Preliminary Remarks, J. Laryng. & Otol. **44**:531-548 (Sept.) 1939.

30. Strauss, H.; Landis, C., and Hunt, W. O.: Acoustic Motor Reactions, Arch. Otolaryng. **28**:941-945 (Dec.) 1938.

high speed motion pictures to study these reactions, found that they all had a similar pattern, which consisted of shutting of the eyes, characteristic distortion of the features, forward movement of the head, raising and drawing forward of the shoulders, abduction, forward elevation and inward rotation of the upper parts of the arms, bending of the elbows, pronation of the forearms, clasping of the hands, contraction of the abdomen, forward movement of the trunk and bending of the legs at the hips and knees—in other words, a reaction of self defense. The authors were particularly interested in the cochleopalpebral phase and found it all was present except in totally deaf persons, certain persons with epilepsy, psychotic patients and those who were unconscious.

Another article on the threshold of unpleasantness of sound in the normal and hard of hearing was presented by Ansberry,[31] who confirms the findings of Fowler, Reger and others that the threshold of hearing in deficient ears is critical. He illustrates the fact that the average of usable sound for persons with subnormal acuity is greatly reduced as their threshold of sound approaches the threshold of unpleasantness. This fact makes it difficult for some deaf persons to employ a hearing aid. When some sounds are amplified enough to be heard, others have become loud enough to be unpleasant.

A study of hearing acuity was made by Kelley[32] on 88 people between 50 and 80 years of age. He grouped his audiometric tests into ten year periods. These showed auditory loss in the tones 2048 and up for all ages, but increasing with each decade. All of these persons heard well up to 512 cycles. This substantiates our knowledge of the loss of acuity for the high tones after the age of 25 to 30 years. The perception of tone was not apparent to persons with presbycusis, but the complex tones probably lacked brilliance. The recognition of speech confirmed the audiometric reading. The ability to hear consonants is lost before the ability to hear the vowels.

Evidence is presented by Harms and Malone[33] which suggests that loss of hearing acuity during the period of speech formation is a cause of stammering. Therefore, the hearing should be tested when one is attempting to determine the cause of the defect in speech. When defects in hearing are found they should be treated by removal, if possible, of any pathologic condition causing the loss in hearing. Hearing aids

31. Ansberry, M.: Auditory Threshold of Unpleasantness in Normal and in Hard of Hearing Subjects, Arch. Otolaryng. **28**:954-958 (Dec.) 1938.

32. Kelley, N. H.: Study in Presbycusis: Auditory Loss with Increasing Age and Its Effect on the Perception of Music and Speech, Arch. Otolaryng. **29**: 506-513 (March) 1939.

33. Harms, M. A., and Malone, J. Y.: Hearing Acuity and Stammering, Ann. Otol., Rhin. & Laryng. **48**:658-662 (Sept.) 1939.

should be used in the correction of speech in these persons if the hearing cannot be improved to a useful level.

One of the principal aims of an investigation on the hearing of school children undertaken by the United States Public Health Service has been the determination of the mode of onset and the degree of progression of the impairment of hearing in childhood. For this purpose periodic audiometric examinations have been made on the same group of children. In 1931 a group of 1,400 school children in Washington, D. C., were given otoscopic examinations and examinations of the nose and throat and tests for acuity of air and bone conduction hearing with a Western Electric 2-A audiometer. Ciocco [34] reports on the reexamination of the air conduction acuity of 552 of these children after an interval of five years.

In the group as a whole the frequency of loss of acuity of hearing for high tones increased by about 300 per cent, while that of impaired perception for the tones of the speech range increased by only about 20 to 30 per cent. In about one fourth of the subjects with a slight degree of impairment as defined, an impairment of greater degree developed, but of the same type, i. e. either loss of acuity for high tones or loss for tones of the speech range.

In the majority of subjects in whom a marked degree of impairment, as defined, was present at the first examination, the same condition was found five years later. The development of impairment involving in particular the tones of the low and middle range does not seem to be related either to the sex of the child or to the age at the first examination. The relative number of children in whom a slight loss of acuity for high tones developed increased concurrently with the age of the children at the first examination. On the other hand, the percentage of the children in whom a marked loss of acuity for high tones developed increased with the age up to 10 years and decreased thereafter. The percentage of boys who acquired a marked loss of acuity for high tones during the interval was three times that of the girls. The remaining forms of auditory defects developed with about equal frequency in the two sexes.

Hill [35] points out that the upper limit of hearing is important in the diagnosis and prognosis of certain types of hearing. An accurate determination by bone conduction is of definite value to the otologist. The monochord is the best available device for measuring the upper tonal range, both by air and by bone conduction. It should, of course, be

34. Ciocco, A.: Audiometric Studies on School Children, Ann. Otol., Rhin. & Laryng. **47**:926-937 (Dec.) 1938.

35. Hill, F. T.: The Use of the Monochord in Routine Tests of Hearing, Laryngoscope **49**:666-677 (Aug.) 1939.

remembered that the monochord is a qualitative instrument and that no single instrument or test is alone sufficient to enable one to make a differential diagnosis of the various types of deafness. Hill said he routinely used the monochord, together with tuning forks and the audiogram, in all his functional tests of hearing.

There is no question that the monochord is preferable to the Galton whistle and the Koenig bars in the measurement of the upper tone limit.

The level of normal hearing by bone conduction in young adults has been carefully worked out by Greenbaum, Kerridge and Ross.[36] They examined 100 people (74 male, 26 female) between the ages of 18 and 25 who had never had any aural disease. Their hearing was carefully measured by two audiometers in a sound-proof room. The authors summarized their results to make a new line for "normal bone conduction" on the ordinary audiometer chart. This new curve is less bumpy than the previous normal curve, which had been based on only about 30 young adults. This new curve, of course, represents the mean. In addition, these authors prepared curves showing the range in which, first, 68 per cent and, secondly, 99 per cent of the values fell. Their results show that a rather wide variation is obtained in testing intelligent and cooperative adults. Their results emphasize the fallacy of labeling bone conduction as abnormal when a determination on a patient is compared with a measurement on a single normal person.

A new type of hearing test similar to the Rinne test is described by Hulka.[37] The end of the bone conduction receiver of the Western Electric 2-B audiometer is placed against the tragus, and this cartilaginous organ is forced into the meatal orifice. This arrangement renders the test different from the routine test for air conduction. The vibrations of the instrument are primarily transmitted by direct contact to the cartilaginous and cutaneous tissues around the meatal opening. From here they are propagated through the air enclosed in the meatus and through the soft and bony tissues of the skull. The closed cavity of the meatus inevitably resembles a sounding box in which the impact of sound waves against the tympanic membrane is reenforced by the reflected portion of the waves. This test the author terms the "tragus test." Its character was tested on 42 ears. Hulka presents the history of 21 cases, together with forty-six audiograms. All ears were tested for air conduction and bone conduction and were given the tragus test. the Gellé test was done in all frequencies of the audiogram.

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36. Greenbaum, A.; Kerridge, P. M., and Ross, E. J.: Normal Hearing by Bone Conduction, J. Laryng. & Otol. **54**:88-92 (Feb.) 1939.

37. Hulka, J. H.: Testing of the Transmission Apparatus with the Bone-Conduction Receiver, Ann. Otol., Rhin. & Laryng. **48**:1020-1054 (Dec.) 1939.

The study shows that the tragus curve for normal ears is situated between the air conduction curve and the bone conduction curve and that the patient's age has an influence on the tragus curve similar to that on the other two curves. With an obstructive lesion the tragus curve is depressed toward the bone conduction curve.

The level of the tragus curve as related to the level of the bone conduction curve was found to vary with the condition of the mucosa of the middle ear. An osseous block in the oval window reduced the hearing in the tragus test more than a fibrous fixation of the stapes. A theory is expressed that the threshold vibrations in the tragus test reach the annular ligaments through the membranous lining of the middle ear, and the tragus conduction was designated as having a membranous character.

A group of ears was studied which in low tones showed crossing of the air conduction curve below the tragus curve. This tragus–air conduction curve crossing was interpreted as ankylosis of the ossicular joints. Attention is drawn to frequent nasopharyngeal involvement in cases of catarrhal deafness and to the similarity of the reactions to the tragus test and the Rinne test.

VESTIBULAR PHYSIOLOGY

Hyndman [38] gives a complete abstract of the literature from 1842 to date. He discusses freely the varied hypotheses and experimental evidence presented, so that one may better understand and appreciate the vestibular labyrinth and its relation to the bony mechanism as a whole. This worth while article should be read by any one wishing to review the subject.

Further experimentation has been carried out to determine the origin of the quick component of labyrinthine nystagmus. Spiegel and Price,[39] operating on the floor of the fourth ventricle in cats which were under anesthesia obtained by the use of pentobarbital sodium, made punctures of the reticulate substances and the raphe of the rhombencephalon. The authors observed the ocular reactions following stimulation of the labyrinth and observed particularly the restitution of function. The suppression of the rhythmic reaction found in the early experimental stage subsides a few days after operation. These observations seem to support the view that the reticulate substance is not indispensable in the mechanism of nystagmus and that the origin of the rhythm must be in the vestibular nuclei.

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38. Hyndman, O. R.: Physiology of the Vestibular Labyrinth, Arch. Otolaryng. **29**:759-819 (May) 1939.

39. Spiegel, E. A., and Price, J. B.: Origin of the Quick Component of Labyrinthine Nystagmus, Arch. Otolaryng. **30**:576-588 (Oct.) 1939.

More investigations have been performed on guinea pigs to determine their reaction to galvanic stimulation. Buchanan and Ladd [40] checked the reactions of 30 animals and confirmed the previous observations that the direction of the reaction is always away from the cathode and toward the anode. The strength of the reaction is dependent on the amount of current used. Occasionally, the authors found, a normal animal would react in the wrong direction, and unilaterally labyrinthectomized animals were predisposed to such reactions, which were about three times more frequent on the sound side than on the labyrinthectomized side. When both labyrinths were destroyed these paradoxic responses were decreased. More current was needed on the side operated on than on the sound side. Reactions were obtained when both labyrinths were removed, indicating that the reaction is probably due to a stimulation of the brain substance itself and not of the labyrinth alone.

In view of the fact that the reactions are not constant, it does not seem justifiable to accept the test for clinical purposes.

Skoog [41] produced an allergic reaction in guinea pigs by injecting sheep-hemolytic rabbit serum into the right carotid artery. This resulted in a symptom complex composed of disturbances of equilibrium and vestibular ocular symptoms. With the aid of intravitam staining with acid dyes, Skoog detected a change in the capillary permeability in the brain stem. He found the affected center to be in the vestibular ganglions of the medulla. He feels that initiation of the symptom complex is independent of the peripheral labyrinth or of the cerebellum. However, this is not taken to mean that one can exclude the possibility that in man the peripheral labyrinth takes part in a similar manner. There may be allergic sensitivity of the tissues in the human labyrinth, for in some persons Ménière's syndrome can be elicited allergically.

Salem [42] says that the otorhinolaryngologic clinic of the school of naval aviation in Rio de Janeiro has about two hundred records pertaining to examinations of the labyrinth of aviators. In the course of these examinations and of continuous control tests on aviators, it was found that life as an aviator rapidly fatigues the organism and influences the vestibular apparatus. The more active the pilot is, the weaker become his vestibular reactions. The rotatory test of Bárány was employed by the author only during the first examination; for experi-

40. Buchanan, A. R., and Ladd, L. D.: The Galvanic Reaction in Guinea Pigs, Arch. Otolaryng. **29**:124-135 (Jan.) 1939.

41. Skoog, T.: Vestibular Syndrome Induced in Guinea Pigs by Allergic Reaction, Acta oto-laryng., 1939, supp. 32, pp. 1-62.

42. Salem, W.: Interpretation of Examination of Labyrinth in Aviators, Presse méd. **47**:1189-1204 (Aug. 2) 1939.

enced aviators he found the test of little value. He regards the caloric test as the best mode of examination of the labyrinth. Aviators acquire a sense of equilibrium superior to normal. But their labyrinthine sensitivity is nearly always diminished; this seems to be a phenomenon of adaptation. By itself hypoexcitability does not reveal any disorder of the labyrinth and has no value in testing aviators. The author also mentions a case of labyrinthine hyperexcitability detected by caloric tests in a stunt flier. This man was permitted to continue ordinary flying but was advised not to do stunt flying and flying at high altitudes.

It is reassuring to find that Maybaum [43] feels that vestibular tests are of considerable assistance in the diagnosis of lesions of the brain stem and particularly in the localization of expanding intracranial lesions. The vestibular and hearing tests are useful in the differential diagnosis of peripheral and central lesions of the eighth nerve and in the differential diagnosis between infratentorial and supratentorial lesions. Maybaum found the caloric test to be much more accurate and reliable than the turning test. The vestibular test is an aid in localizing the lesion but does not determine its nature. It is essential that the tests be performed by a standardized technic. The proper evaluation of any test depends on the experience of the examiner. In the author's hands, hearing and caloric tests have in some 40 verified cases aided in the definite localization of lesions of the posterior fossa. Tests not only aid in the localization, by exclusion, but also enable one to make an early diagnosis of tumors of the cerebellopontile angle. Vestibular-cochlear observations help in evaluating Ménière's syndrome.

Ohma [44] studied sections of the temporal bones in 200 persons ranging in age from 2 days to 86 years, with reference to the fluid spaces of the labyrinth. In 2 specimens he found the aquaeductus cochleae to be obliterated, but the membranous labyrinth was unchanged. The author felt that the distended spiral veins had assumed the function of the aquaeductus. In 3 cases marked stenosis of the aquaeductus cochleae was associated with a collapse of Reissner's membrane and collapse or ectasia of the sacculus. Ohma felt that this resulted from a disturbance in the pressure equilibrium between the perilymph and the endolymph. In 3 instances in which the ductus endolymphaticus was partially occluded there was ectasia of Reissner's membrane, the sacculus, the ductus reuniens and the portions of the ductus endolymphaticus directly above the occlusion. The saccus endolymphaticus alone was collapsed. The writer concludes that the perilymph as well as the endolymph,

43. Maybaum, J. L.: Value of the Vestibular Test in Neurological Diagnosis, Ann. Otol., Rhin. & Laryng. **48**:484-490 (June) 1939.

44. Ohma, M.: Relationship of Morbid Disturbances of the Labyrinthine Fluid Spaces and Canals, Hals-, Nasen- u. Ohrenarzt (pt. 1) **30**:73-87 (March) 1939.

originates in the labyrinth, although he does not state where the site is located. Perilymph normally passes out through the aquaeductus cochleae but may also be absorbed by the veins, especially those in the scala tympani. Endolymph is probably absorbed by the epithelium of the saccus endolymphaticus and the ductus endolymphaticus. Disturbances of flow through the endolymphatic system lead to equilibratory disturbances between endolymph and perilymph. Ectasia of the saccus endolymphaticus results. The "utriculoendolymphatic valve" of Bast enables the endolymph pressure in the semicircular canals and utricles to be maintained independently of the pressure in the sacculus and ductus cochlearis.

It must be stressed that time of the removal of the specimen and the type of fixation used greatly modify postmortem appearances, so that conclusions drawn are always open to doubt.

In a previous article Hallpike and Cairns [45] gave a description of the pathologic observations in the temporal bones of 2 typical subjects with Ménière's syndrome. Certain peculiar changes were disclosed in the labyrinth, and the changes were considered to support the view that the disorder was due to a specific disease in the labyrinth itself. The pathologic changes in the temporal bones of a third subject, examined by Hallpike and Wright,[46] are reported. In the previous report the view was advanced that the primary mechanism of the disease was an obstructive distention of the endolymph system. This distention occurred in the present case. The peculiar nature of the changes makes it reasonable to regard them as constituting the essential morbid anatomy of a specific disease of the labyrinth. The relatively slight degree of the endolymphatic distention in the present instance and the absence of changes in Corti's organ or in the stria vascularis, such as were disclosed in both of the ears previously reported on, are considered in greater detail. In both particulars it seems likely that the principal factor involved was the short duration of the disease.

Talbott and Brown [47] conclude that neither hydration or alkalosis nor an elevated serum sodium content is a necessary accompaniment of acute symptoms of Ménière's syndrome. During eighteen months patients suffering from this malady were treated by a high intake of potassium, meanwhile being allowed an otherwise normal diet. The therapeutic effect of this regimen was reassuring. It cannot be con-

45. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625-654 (Oct.) 1938.

46. Hallpike, C. S., and Wright, A. J.: Histologic Changes in Temporal Bones of Cases of Ménière's Disease, Proc. Roy. Soc. Med. **32**:1515-1722 (Oct.) 1939.

47. Talbott, J. H., and Brown, M.: Ménière's Syndrome: Acid-Base Constituents of the Blood; Treatment with Potassium Chloride, J. A. M. A. **114**:125-130 (Jan. 13) 1940.

sidered as a cure for all the symptoms, but clinical improvement was impressive. Since the institution of this regimen, surgical treatment had not been carried out in any case.

Caisson disease, or the compressed air disease, affects the internal ear as well as other organs. It is during the stage of decompression that the most damage can occur. Crespi Reghizzi,[48] in a study of caisson disease, found that labyrinthine lesions are fortunately rare. When they do occur it is difficult to give any prognosis. Usually recovery is gradual and incomplete. Vertigo, deafness and tinnitus may practically disappear, or any one, or all, may remain permanently. This, of course, depends on the extent of the damage to the labyrinth.

Chiappe[49] experimented on guinea pigs, subjecting them to compression and decompression at different rates and intervals. Histologic examination showed the gravity of the lesion to depend on (1) the pressure, (2) the duration of the compression and (3) the rate of decompression. The third factor is the most important.

The minimum effect of decompression on the internal ear is merely one of vasomotor congestion. More marked effects result in traumatic degenerative lesions. Inflammatory changes are often demonstrated.

When the decompression rate is rapid the effects are severe. A traumatic labyrinthitis results from infiltrating hemorrhages. These are both endovascular and perivascular in type. They may cause destruction of the ganglions of the cochlear and vestibular nerves.

It is pointed out by Fowler[50] that there are basically two types of tinnitus. First, there is the vibratory, or peripheral, type. This is caused by actual vibrations from any source reaching the end organ of the cochlea. It may at times occur in normal ears and can be self induced. It is easily masked by extraneous sounds because it is set up by actual vibrations reaching the cochlea. Vibratory tinnitus is made up predominantly of the lower frequencies.

Secondly, there is the nonvibratory tinnitus, which may be either peripheral or central. It is caused by factors other than vibratory, such as biochemical stimulation. It is wholly due to lesions of the neural mechanism of hearing. Changes of circulation in the cochlea due to general vasomotor disturbance, local pressure and variations in blood chemistry anywhere along the neural pathway may cause tinnitus, independent of any cochlear vibrations. There is usually a predominance of high-pitched sounds. Disturbances of the gastrointestinal

48. Crespi Reghizzi, A.: A Clinical Study of Caisson Disease, Arch. ital. di otol. **51**:408-420 (Aug.) 1939.

49. Chiappe, E.: Lesions of the Internal Ear Caused by Decompression, Oto-rino-laring. ital. **9**:149-178 (March) 1939.

50. Fowler, E. P.: Head Noises and Deafness: Peripheral and Central, Laryngoscope **49**:1011-1024 (Oct.) 1939.

tract, smoking, temporomandibular dysfunction, etc., may initiate this type of tinnitus. The central tinnitus is masked with greater difficulty than the peripheral. This is because the masking sounds set up in the end organs do not get through to the central areas. The lesions have caused a block in the neural pathway. According to Fowler, it is therefore possible to use tinnitus itself as a means to differentiate between central and peripheral tinnitus and deafness.

This is open to question.

Hayden and Chainski [51] discuss the etiology of tinnitus and sum up the recent literature on the treatment of the condition. They give a particularly good account of the various forms of medical treatment which have been employed. Surgical treatment is unsatisfactory. No one type of treatment is satisfactory in all cases. It requires a diligent and systematic search on the part of the physician to discover a satisfactory method of treatment in the individual case, and in certain cases the condition must be tagged "incurable."

51. Hayden, D. B., and Chainski, E. L.: Tinnitus: Etiology and Evaluation of the Various Methods of Treatment, Ann. Otol., Rhin. & Laryng. **48**:443-453 (June) 1939.

Abstracts from Current Literature

Ear

SEPTIC THROMBOSIS OF THE JUGULAR BULB. A. ROSSI, Arch. ital. di otol. **51**:513 (Oct.) 1939.

Rossi reports 2 cases of thrombosis of the jugular bulb secondary to thrombosis of the lateral sinus and 1 case of primary thrombosis of the bulb. Thrombosis of the jugular bulb is more frequent than is generally supposed. This opinion is supported by anatomic investigations, which demonstrate the constant presence of venous emissaries from the surrounding structures; it is supported also by the known relations of the bulb to the hypotympanum and the mastoid cells. The importance of early recognition of the condition, followed by complete drainage and resection of the jugular vein is stressed. Azo dyes, bacterial vaccines and supportive measures after operation are recommended.

DENNIS, San Diego, Calif.

TREATMENT OF ACUTE SUPPURATIVE OTITIS MEDIA WITH ALCOHOL IRRIGATIONS. ARNOLD JORGENSEN, Pract. oto-rhino-laryng. **2**:261 (Sept.) 1939.

The author treated a large series of patients having acute suppurative otitis media by means of mass irrigations with a 70 per cent solution of ethyl alcohol four to six times a day, using from 20 to 60 cc. of the solution. He found that the canal was readily cleansed of any secretions and debris and that the irritated or eczematous condition of the external canal, so commonly found in association with the acute suppurations, rarely occurred. Hemorrhagic bullae were quickly dried; the paracentesis openings (or spontaneous perforations) always remained open, while granulations and nipple-like swellings (or protrusions of mucous membrane through the tympanic membrane) cleared up readily. He states, however, that when a "nipple" persists for a definite period a mastoidectomy is usually unavoidable. But the pain associated with the treatment is rather marked. This is true of infants and children. The pain usually becomes more tolerable after repeated irrigations.

He reports on 2 series of patients: (1) a group of 357 patients treated by alcohol irrigations and (2) a control series of 410 patients treated by the ordinary water irrigations. In general, while the results show some minor variations in the two groups, these are not particularly significant.

His summary gives the advantages of the treatment as follows: 1. The number of mastoidectomies decreased by 10 per cent. 2. The perforation is usually kept open. 3. Eczema of the external auditory canal is uncommon. 4. Because of the widely patent opening in the tympanic membrane, the alcohol may penetrate into the middle ear and so have beneficial influence on both this space and the eustachian tube. However, he cites certain disadvantages of the treatment: 1. Hospitalization, he believes, is necessary for the proper carrying out of the treatment. 2. The treatment is rather painful. 3. While the incidence of mastoiditis may be decreased, the discharge from the middle ear usually persists for a longer time than when the ordinary therapeutic methods are used.

PERSKY, Philadelphia.

DEVELOPMENT OF THE TYMPANIC CAVITY AND THE ROLE OF INFECTION OF THE MIDDLE EAR. LUZIUS RÜEDI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:175, 1939.

The author accepts the Wittmaack principle that a condensation or sclerosis of bone follows an inflammatory hyperplastic change of the mucous membrane

in either intrauterine or extrauterine life. As a result of this infantile otitis media, there occurs a hyperplastic alteration of the mucous membrane, with a subsequent sclerosis of bone. Assuming this dictum, Rüedi believes that these early infantile hyperplastic conditions cause a retardation in the pneumatization of the mastoid process and that, since there is a hyperplastic (and potentially sclerotic) state, chronic suppurations of the middle ear can develop readily. The epithelium of the first and the second pharyngeal pouch ordinarily becomes connective tissue and bone of a "preformed" middle ear space. There follows an active, space-forming protrusion of the mucous membrane of this middle ear, which, under the guidance of the epithelium, continues into the expanding temporal bone.

Normal pneumatization of the temporal bone occurs in three periods: (1) up to the end of the first year, development of the tympanic cavity and an antrum; (2) from the second to the third year, development of the fully pneumatized cells into the mastoid process, and finally (3) the period in which pneumatized cells expand with the growing temporal bone until the entire bone is fully pneumatized. Every alteration from a typically normal course of pneumatization would depend on an inflammatory damage to the mucous membrane which is concerned with this space building process, and hence any limitation in pneumatization is definitely pathologic.

Rüedi made a study of the tympanic space and the mastoid process in a group of patients whose ages ranged from the fifth embryonal month up to the tenth year and, as a result, believes that pneumatization depends primarily on a "preformation" of cells in the temporal bone. The development of this bony space is independent of the thickness of the mucous membrane, which may either line or fill the space. There are two processes that can be differentiated: (1) the "preformation" of the bony spaces, which begins with an early embryonal connective tissue, bony differentiation into a tympanic cavity and an antrum, and which proceeds after the seventh embryonal month to the enlargement of these bony spaces and at the same time the beginning of a development of a cell system which expands into the temporal bone; (2) the "air space building," characterized by an increase in the air capacity of the epithelial sacs within the "preformed" bone spaces. While these two processes begin about the same time their origin is at different sites, and the space between these two sites is filled in by a loose embryonal connective tissue. Rüedi found that at the end of the first year the air spaces are usually filled out and the embryonal connective tissue has been completely eliminated both between the cells of the bony tissue and between the air spaces. In an inflammatory condition in newborn and older infants, there develops a disproportion between these two processes, which is particularly emphasized by a retardation of the second phase, the so-called air space building. In consequence, there is a variable residuum of embryonal connective tissue, which depends on the degree of interference in the air space development. Rüedi terms this difference in the rhythmic construction of the mastoid process a dissociation of the space building process.

Another subject considered is the occurrence of adhesive processes in the course of the development of a mastoid. These may be present with extensive pneumatization or a frank dissociation in the space building process. Rüedi believes that this picture is usually due to variation in the mastoid development within normal limits, and he usually finds that these adhesive processes are present in well developed mastoid processes. In conditions of chronic inflammation of the middle ear these usually occur as a dissociation of the building process and as a result of the residual embryonal connective tissue. He believes further that this residual embryonal tissue can find a fertile soil in the epitympanic space and readily lend itself to the formation of cholesteatoma, and moreover that there is also a resultant sclerosis of the mastoid process due to a retardation of the "preformed" bony state. This probably takes the form of a productive osteitis. Since this new bone is poor in bone marrow, its structure is denser.

The degree of sclerosis depends more or less on the extent and development of the "preformed" cells at the time of onset of the chronic suppuration.

The author makes first the following theoretic conclusions: 1. The development of the tympanic and mastoid spaces occurs as the result of two different processes, a bony space "preformation" and an air space building. Both processes begin simultaneously at different sites, the intervening space being filled by an embryonal type of connective tissue. Normally, their progress is an approximation of these two processes, terminating at the latest toward the end of the first year of life (association). 2. Infants who are affected by an acute inflammation usually show a slow expansion in the air space building. The rate of approximation of the two processes is disturbed, and, with the retardation of the air space building, a dissociation occurs. After the subsidence of the acute inflammation, the middle ear and mastoid will heal. Pneumatization may continue, and more or less adhesive processes will be present. 4. Small children with thickening of the embryonal connective tissue, due to chronic inflammation resulting from the dissociation of the building process, will ultimately present a favorable site for the development of an epitympanic cholesteatoma. 5. Since both processes of bone development are affected by chronic inflammation, sclerotic bone will result.

Secondly, he draws some practical conclusions: 1. Since the course of these space building processes cannot be controlled in the living, all infants should be guarded against recurrent acute infections. 2. There are no definite, specific factors that are known to influence the rate of development of these processes. Crowe and Guild's theory, he suggests, of applying radium to the orifice of the eustachian tube in some of the children who are hard of hearing, may cause shrinkage of the submucous tissue and so permit a leveling out of the dissociation processes. 3. Cholesteatomas, with small tympanic perforations, particularly in Shrapnell's membrane, should be operated on early, since cholesteatoma rarely yields to conservative treatment and, owing to a continuation of the dissociation process, grows and often leads to intracranial complications.

PERSKY, Philadelphia.

ROENTGENOGRAPHIC STUDY OF REGENERATION OF BONES IN MASTOIDITIS. R. MITTERMAIER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:214, 1939.

The author made a roentgen study of bone regeneration of the mastoid process to determine: 1. Can bone regeneration be recognized roentgenologically? 2. How can this be gaged?

He states that, roentgenologically it is not possible to differentiate bone regeneration as to whether it originated from the periosteum or from the bone marrow proper. He studied a series of cases of both acute and chronic mastoiditis (with and without operation) over a period of years and made the conclusion that regeneration of bone is an inflammatory reaction. The degree of initial irritation usually controls the extent of reaction. For example, a mild inflammatory infection will produce only a small amount of bone regeneration, and vice versa, when the infection is marked, there occurs an extensive regeneration of bone, which at first is spongy and later becomes sclerotic. Roentgenologically it is difficult to identify this osseous regeneration. There may be sclerosis after a cavity formation, which may be due, on one hand, to a limited pneumatization or, on the other hand, to a secondary ossification. This differentiation is impossible by means of a single roentgen examination, but it may be deduced by repeated studies over a number of years. The author believes further that structural change in the regenerated bone can occur, so as to resemble a partial pneumatization of the given area.

PERSKY, Philadelphia.

THE BLUE EAR DRUM. ARISTIDES MONTEIRO, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:242, 1939.

The author reports a group of 3 cases of blue ear drum, in the first case associated with a tumor in the neck, in the second with congenital heart disease and in the third with general varicosities of the legs.

After citing a number of cases from the literature, he concludes that this phenomenon can occur as a result of the following conditions: (1) a dehiscence in the floor of the tympanic cavity, with an upward protrusion of the jugular bulb, (2) hemorrhage in the tympanic cavity, (3) gelatinous exudates within the cavity, (4) varices of the ear drum, (5) disturbance of collateral circulation of the middle ear (tumor) and (6) congenital heart disease.

In view of the importance of the first mentioned condition, namely, dehiscences in the tympanic cavity, the author stresses the dangers of paracentesis in the posteroinferior quadrant of the ear drum. PERSKY, Philadelphia.

CONSIDERATION OF THE ANATOMIC STRUCTURES OF THE TEMPORAL BONE AS FOUND AT THE TIME OF OPERATION FOR ACUTE MASTOIDITIS. HELMUTH RICHTER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:266, 1939.

Richter states that the anatomic structure of the temporal bone plays an important role in the course of an acute mastoiditis. The importance of this is apparent, since stasis of any suppuration is the first means or avenue for the development of dangerous intracranial complications. He has shown that extensive, pneumatized mastoid processes favor suppurative necrosis of the cells, while limited pneumatization tends to cause stasis and retardation of pus. Because of this limited pneumatization, it is difficult to identify the empyema, since there is a lack of clinical symptoms, such as postauricular edema, pain and tenderness. Therefore the mastoid process with limited pneumatization is dangerous, especially when an acute exacerbation is superimposed on an old sclerosis. The author advises a careful watch as to the possibility of an intracranial complication.

Richter reports a series of 50 cases of acute mastoiditis in which mastoidectomies were performed, within a four month period, 48 per cent of the conditions occurring within the first and second decades of life. Four patients died, 3 of whom had poor pneumatization. Of the 4, 1 died of sinus thrombosis, poor pneumatization and infection due to a hemolytic staphylococcic organism; the second, of sinus thrombosis, meningitis, poor pneumatization and infection produced by Pneumococcus type III; the third, of extradural abscess and generalized septicemia, but having extensive pneumatization, and the fourth, of cerebral abscess, with poor pneumatization and an infection caused by P. type III. A further analysis was made of the entire group; roentgenograms had not previously been made of 12 of them, and 9 were accepted because of inconsequential etiologic factors. Of the remaining 29 patients, 10 had normal pneumatization while 19 had definitely constricted or limited pneumatization.

In analyzing some of the symptoms, Richter says that he feels that postauricular pain and tenderness are not dependable symptoms, particularly in an older person, or in one with poor pneumatization of the mastoid process. He thinks that they should not be considered as indications for mastoidectomy. He places more importance on the general appearance of the patient, the duration of symptoms and the otoscopic findings, particularly tinnitus and a drooping of the posterosuperior wall of the external canal.

The bacteria, as found in these cases in which operation was done, is rather interesting in that, while Streptococcus haemolyticus was found equally distributed in the two groups (those with the normal and those with the poorly pneumatized mastoid processes), there was a marked increase of P. type III (mucosus) in the latter group. While Richter is impressed by the frequent occurrence of the P. mucosus in poorly pneumatized mastoid processes, he believes that the slow course of the disease is directly due to the poor development of the mastoid process rather than to the type of organism.

He also believes that, since 21 of the 38 patients examined roentgenographically showed a poorly pneumatized mastoid process, the importance of this roentgenologic finding is not so great from the diagnostic or clinical standpoint. He feels rather that it should be an aid to better gaging and interpretation of the significance of other symptoms.

In summarizing the 50 cases, he draws a relationship between the anatomic structure of the temporal bone and the course of the disease. Poor pneumatization is more dangerous to the life of the patient, since the tendency to stasis of the suppuration (loculated empyema) is greater and the interpretation of the symptoms more difficult. He repeats that the general impression of the patient and a summation of all the general and local symptoms should give the basic indication for operation.

PERSKY, Philadelphia.

A CASE OF ACUTE SUPPURATIVE OTITIS MEDIA DUE TO PNEUMOCOCCUS TYPE II. KIKUJI MARUYAMA, Ztschr. f. oto-rhino-laryng. (Jap.) **45**:703 (June) 1939.

The patient, a 40 year old man, had acute otitis media on Aug. 3, 1937 without prodromal symptoms. The next day otalgia subsided, but impairment in hearing continued. An otologist inflated the eustachian tube without appreciable benefit. On August 24 paracentesis was done, and a small amount of serosanguineous discharge was evacuated. For seven days there was a varying amount of drainage; then the ear ceased to discharge, the ear drum healed and hearing was restored.

The bacteriologic study of the exudate showed encapsulated diplococci. On the agar culture mediums the organisms produced colonies of thin serous fluid. It fermented insulin and was bile soluble. The organisms gave strongly positive reactions to agglutination and precipitation tests for the type II pneumococcus.

In Wirth's 271 cases of acute otitis media, 118 growths were due to pneumococci, of which 18 lesions were caused by type I, 10 by type II, 54 by type III and 26 by type IV.

Bauer and Clair observed 13 cases of mastoiditis due to pneumococci and there were 2 growths due to type I, 9 to type II and 2 to type IV.

Christiansen stated that intracranial complications of otitic origin are due to Pneumococcus type III.

In the series of 412 cases of acute suppurative otitis in which culture was taken at the time of paracentesis, Ito states that in 212 (51.5 per cent) the disease was pneumococcic in origin. In 20 of these the organisms were type I, in 2 type II, in 44 type III and in the remaining 146 various groups in type IV.

It is evident that acute suppurative otitis media due to Pneumococcus type II is comparatively rare and that its clinical course is mild when it does occur.

HARA, Los Angeles.

A DEMONSTRATION ON MONKEYS OF THE HOLMGREN OPERATION FOR OTOSCLEROSIS. F. R. NAGER, Acta oto-laryng. **27**:350, 1939.

Gunnar Holmgren did his operation for otosclerosis on a number of monkeys, choosing this animal because its labyrinth is similar to that of man. After a variable period the animals were killed, and their temporal bones were removed and sent to Nager for sectioning, in order to determine what happened to the fistula operatively produced. The operation antedated the death of the animal by from twenty-three to three hundred and sixty-five days.

Holmgren had used various substances to keep the fistula open, such as fat, gold foil, transplanted epithelium and radium. Nager found that in all the specimens the fistula was closed by bone originating from the periosteal layer. The perichondral layer showed no reaction, whereas the endosteal layer showed some formation of connective tissue.

In the cases in which squamous cell epithelium had been planted into the wound the production of bone around the edges of the fistula seemed to be less. When radium had been used the production of bone was markedly reduced.

It therefore seemed to Nager that further experimentation with radium applied postoperatively might lead to better results in maintaining a patent fistula.

GROVE, Milwaukee.

Nose

ALLEGED PAINFUL SEQUELAE AND POOR RESULTS OF ANTROSTOMY. A. B. K. WATKINS, M. J. Australia **1**:302 (Feb. 25) 1939.

The reasons for the bad repute into which antrostomy has fallen in the minds of both physicians and lay people are discussed under the following headings: (1) operations performed in unsuitable cases, (2) unsuitable operations, (3) other nasal lesions which have been overlooked and (4) technical failures in operation.

GONCE, Madison, Wis. [AM. J. DIS. CHILD.]

PROGRESSIVE OSTEITIS OF THE FACE, REFRACTORY TO SURGICAL MEASURES AND CURED BY SULFANILAMIDE. P. JACQUES, Oto-rhino-laryng. internat. **23**:465 (Sept.) 1939.

This article is a case report of a man, aged 39 years, who suffered with osteomyelitis of dental origin, involving the superior maxilla. In spite of repeated operations, extending over several months, the patient's condition became grave and the infection continued to spread. At this point sulfanilamide was administered by mouth, and marked amelioration was obtained after three weeks, followed by complete recovery at the end of six weeks. DENNIS, San Diego, Calif.

EVALUATION OF ROENTGEN DIAGNOSIS OF MAXILLARY SINUS DISEASE. F. J. MAYER, Monatschr. f. Ohrenh. **73**:651 (Oct.) 1939.

In 3 cases roentgenographic findings were described as normal, while irrigation of the antrum revealed purulent secretion. Therefore, it is advisable to irrigate the antrum if clinical findings indicate, even in the face of normal roentgenographic appearances. The author advises puncture of the inferior meatus and the use of isotonic solutions, which render irrigation harmless. LEDERER, Chicago.

MEDIAL ORBITAL CONDUCTION ANESTHESIA OF THE ETHMOID NERVE IN ETHMOID SURGERY. F. J. MAYER, Monatschr. f. Ohrenh. **73**:657 (Oct.) 1939.

To obtain a satisfactory anesthesia in operations on the ethmoid region, the transorbital injection of the ethmoid nerves is indicated. In 150 cases in which this type of anesthesia was used, hematomas occurred in 4 cases and cursory amauroses in 5. All of the amauroses subsided after a few hours. The fundi were found to be normal, so that the cause was attributed to a retrobulbar lesion of the nerve. No further damage to the eye or orbit was noted; in spite of these accidents, therefore, the transorbital anesthesia, according to Mayer, is not contraindicated.

LEDERER, Chicago.

TRANSANTRAL ETHMOIDECTOMY (MODIFICATION OF METHOD OF ERMIRO DE LIMA). PAULO MANGABEIRA-ALBERNAZ, Pract. oto-rhino-laryng. **2**:352 (Nov.) 1939.

The author, following the Ermiro de Lima technic, performs a Caldwell-Luc operation; then he locates the natural ostium and makes an incision both behind and in front of the ostium. He raises the mucous membrane, enters the ethmoid labyrinth and completely exenterates this region. The anterior angle between the ethmoid bone and the lamina papyracea serves as a guide to the nasofrontal duct and the frontal sinuses. Working posteriorly along this ethmoid capsule, he enters the sphenoid readily.

He believes that this is a simple procedure for the complete evacuation of the ethmoid and sphenoid sinuses and that it permits a ready drainage of the frontal sinuses.

PERSKY, Philadelphia.

Miscellaneous

WEATHER CONDITIONS AND ASTHMA. Current Comment, J. A. M. A. **113**:1036 (Sept. 9) 1939.

Investigators have been able to produce distinct asthmatic symptoms in guinea pigs by making them breathe a spray of a "vapor" to which these animals were known or proved to be sensitized. At necropsy, when death resulted, in several of the experiments, the classic appearances of acute anaphylactic shock were observed.

By varying the richness of the spray, recording from minute to minute the severity of the asthmatic attack and using a sufficiently large number of animals, a reliable mathematical expression of the average group severity was found.

It was found that the statistical severity of the "vapor" asthma is not dependent on such meteorologic factors as temperature, humidity, or atmospheric pressure as long as these factors remain constant on the day of the experiment, but during rapid changes in the weather the average severity increased about 50 per cent.

The new field of experimental meteorobiology may assume an important role in future therapeutic study.

GORDON, Philadelphia.

USE OF VINYL ETHER (VINETHENE) IN INFANCY AND CHILDHOOD (REPORT OF 100 CASES). R. E. GROSS, New England J. Med. **220**:334 (Feb. 23) 1939.

The type of case in which vinyl ether anesthesia is particularly useful is often encountered in the work of outpatient departments or in the pediatrician's practice.

Vinyl ether (vinethene) was employed as a general anesthetic in a series of 100 infants and children ranging from 1 month to 11 years of age. It was found extremely satisfactory for minor operative procedures, and in no case was there any alarming or untoward reaction accompanying or following the use of it. The extremely rapid induction of anesthesia by this drug permitted full muscular relaxation in thirty or forty-five seconds. The period of recovery was likewise short, and in practically all cases the patient had regained consciousness in two or three minutes; the older children were talking and sitting up by the end of this time. There was postanesthetic vomiting in only 5 cases. The drug can be given easily and quickly on an open mask, and when thus employed in the present series it was always satisfactory.

Vinyl ether appears to be the safest and most satisfactory general anesthetic yet produced for operative procedures of five or ten minutes' duration in infancy or childhood. The cost of the anesthetic is moderate, ranging from 25 to 40 cents in the average case.

GENGENBACH, Denver. [AM. J. DIS. CHILD.]

THE TREATMENT OF LEUKOPLAKIA BUCCALIS AND RELATED LESIONS WITH ESTROGENIC HORMONE. IRA T. NATHANSON and DAVID B. WEISBERGER, New England J. Med. **221**:556 (Oct. 12) 1939.

The authors have noted that leukoplakia of the mucous membrane is not uncommonly associated with disturbance in the menstrual cycle and particularly with the menopause. Similar lesions of the vulva, vagina and cervix occur in the same age group; therefore, it seemed possible that these analogous histologic abnormalities were associated with the same basic systemic factors.

Included in this report are 38 patients with leukoplakia buccalis, of whom 25 were women and 13 were men. The areas most commonly involved, arranged in

the order of their frequency, were the mucous membrane of the cheek, particularly in the molar region, the tongue, the floor of the mouth and the palate.

The treatment of the condition was by the use of estrogen, based on the premise of "sex hormone" deficiency. The substance was administered in two forms. Estradiol benzoate was used subcutaneously and with this alpha estradiol was given orally. In the second group oral medication of alpha estradiol was used.

The results of the treatment were as follows: complete disappearance of the lesions in 42 per cent of the cases; marked improvement in 39 per cent, and no change in 7 per cent. Results were the same with both forms of treatment; however, administration by the subcutaneous route produced results in about one-sixth the time taken to produce them by oral administration.

Because of a reappearance of the leukoplakia in three to six months after therapy was discontinued, it has been suggested that a maintenance dose of the drug be used. This maintenance dose may be given daily by mouth with satisfactory results.

SCHALL, Boston.

SEA SICKNESS. LARROUDÉ, Oto-rhino-laryng. internat. **23**:513 (Oct.) 1939.

The mechanism of the production of seasickness is just beginning to be understood. Actually there are a number of forms of seasickness, varying with the person affected. Among all the theories, one fact is outstanding: The cause of seasickness in a person who has not previously experienced the condition is the movement of the boat. Naupathia is a disease of equilibrium. The parts involved in maintaining equilibrium are the peripheral and deep sensory nerves, the nerves of vision and hearing, which aid orientation, and the peripheral apparatus of static and dynamic equilibrium, situated in the posterior labyrinth, viz. the utriculosaccular system, which presides over the position of the head in space, and the apparatus of kinetic sense, formed by the semicircular canal system. The movements of a boat at sea may be resolved into pitching, rolling and vertical movements produced by the swells. The greater role is played by the otolithic apparatus because it is more easily excited, a heavy sea causing excitations amounting to fifty times the value of normal excitations, while angular displacements determine excitations only double the perceptible minimum. Simple rolling of the ship is much less irritating to the otolithic system because it affects chiefly the saccular otoliths, which are antagonistic to each other. The influence of disturbances of other elements of equilibrium in the genesis of naupathia is also important. The rapid movements and changes of position disturb profoundly the function of synthesis of the varied sensations received by the nerve centers. The disturbed sense of vision also plays a minor part, although blind persons do become seasick. Other contributing factors are imperfect pulmonary ventilation, resulting in the retention of carbon dioxide, disturbing ship odors and psychic conditioned reflexes. Cases of persons becoming seasick even several days before embarking, from recalling the memory of previous experiences, are well known. Equally well known is the effect of fear in inhibiting manifestations of seasickness. The peripheromesencephalic vestibular apparatus constitutes a reflex arc, having over it an automatic regulatory apparatus, the pathways of which unite in the red nucleus. Above this regulatory apparatus lie the cortical centers in the temporal and the parietal lobe, united to those of the frontal lobe by association tracts. The question of the influence of vestibular hallucinations is far from resolved.

The classic symptoms of vomiting, pallor, salivation, bradycardia, vertigo, headache and obnubilation are often accompanied by lowered arterial tension, acidosis and hypochloremia. These symptoms correspond to an abnormal excitation of the neurovegetative system and to biochemical phenomena, the consequences of colloidoclastic shock or a disturbance of the digestive apparatus, such as hepatic insufficiency, chronic appendicitis and spastic colitis. In order to explain the excitability of the neurovegetative system, the influence of dystonia is to be considered. The final result of abnormal excitation of the system of equilibrium is disequilibrium of the vagosympathetic system, which is most manifest in those in

whom there is instability of sympathetic and parasympathetic equilibrium. It is the vagotonic persons who suffer most from seasickness, while in the sympatheticotonic ones the symptoms are much milder. The prolongation of the reflex of seasickness is explained by the increase in the activity of certain glands of internal secretion. The vomiting, respiratory and cardiac centers react to physicochemical changes in their surroundings. Then a reflex action would provoke a humoral modification, which in turn would produce a reflex action—a reciprocal play of actions and reactions.

The best treatment of seasickness is one that blocks the greatest number of centripetal and centrifugal impulses capable of causing it, especially those affecting the vomiting center. The greatest success has followed the use of drugs acting on the sympathetic and parasympathetic systems, particularly the vagus nerve. For obtaining physiologic section of the pneumogastric nerve, atropine is efficacious; for that of the sympathetic nerve, the neutral tartrate of ergotamine is useful. Preventive measures are important if the person can be seen some time before embarking. An examination of the general condition, including the humoral equilibrium, and the institution of measures appropriate to the findings are necessary. Naupathia affects the entire organism. It is a polymorphous disease that may be grouped into a number of types, which means that there is no single remedy for all cases.

DENNIS, San Diego, Calif.

HALLUCINATIONS AND VESTIBULAR FUNCTIONS. LHERMITTE and BINEAU, Rev. d'oto-neuro-opht. **17**:241 (April) 1939.

Bonnier showed that disturbances of the equilibratory apparatus can cause curious modifications of the personality. It is easy to comprehend how false perceptions engendered by disorders of the vestibular apparatus have a special bearing on the consciousness one has of the body, "the image of the corporeal me." Lhermitte and Bineau report 2 cases. The first patient had hallucinations of the corporeal schema and visual hallucinations, accompanied by intense vertigo, nystagmus, vomiting and a sensation of spatial displacement. To the second patient the head seemed not to be an extension of the body but to lie to one side. In both patients a lesion of the central vestibular apparatus was evident, and all symptoms were referable to the rhombencephalon. In Stenvers' case the visual hallucinations were due to a tuberculoma located in the pons, causing gross changes in the central vestibular pathways.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

GRANULOPENIC SYNDROMES ASSOCIATED WITH NECROTIC ANGINA OCCURRING DURING TREATMENT WITH ARSPHENAMINE. G. SAIBENE, Arch. ital. di otol. **51**:458 (Sept.) 1939.

Two cases of a grave toxic syndrome occurring during treatment with arsphenamine are reported. The syndromes were characterized by intense mucocutaneous purpura, by rapidly developing anemia and serious leukopenia involving all the nucleated elements of the peripheral blood and by necrotic angina. The outcome was fatal in both cases. A histologic study of the necrotic tissues revealed extensive destruction in the region of the faucial isthmus, hypopharynx and larynx. It seems beyond question that both arsenic and benzene share in producing the phenomenon. It is suggested that probably a favorable terrain may be created by the syphilitic infection. As a preventive measure, Saibene suggests that before inaugurating arsenical treatment one should make a study of the blood picture to determine if even slight signs of dyscrasia are present and that during the treatment a close watch should be kept for signs of danger, such as petechiae, small hemorrhages and infectious processes, especially the common anginas associated with spirilla.

DENNIS, San Diego, Calif.

MORPHOLOGY OF THE CAPILLARIES IN PERSONS WITH ADENOIDS. C. CALABRESI, Arch. ital. di otol. **51**:485 (Oct.) 1939.

Calabresi studied, with the capillariscope, the subcutaneous capillaries in 65 children with hypertrophy of the pharyngeal tonsil. The instrument was applied to the third finger, and photomicrographs were taken. Because of their general hormonic disequilibrium, persons with adenoids offer a good field for the study of the morphologic and morphogenic behavior of the cutaneous capillaries in comparison with the somatopsychic conditions. Normally the capillaries are arranged in "hairpin-like" loops, which arise from the subcutaneous, horizontal, vascular network and are disposed perpendicularly to the cutaneous surface. In 44 of the subjects the capillary structure was normal; in 21 the picture represented a primitive type of development. In 4 of the latter group, who presented marked signs of the adenoid syndrome with retarded somatic and psychic development, the archicapillary form of development persisted. Sixteen showed persistence of the mesocapillary type, in some of whom neocapillaries were present. One showed classic hypoplasia of the capillaries, with reduction of the caliber and size of the loops. The explanation of the complex physiologic mechanism responsible for these phenomena is difficult. Calabresi believes that the condition is of endocrine origin; the vitiated secretion of the glands of internal secretion plus the dysfunction of the neurovegetative system existing in the adenoid bearer exercises an influence on the development of the peripheral circulatory apparatus, just as they do on the general somatopsychic development. DENNIS, San Diego, Calif.

REPORT OF A CASE OF A TUMOR ORIGINATING FROM THE FACIAL NERVE. A. REJTÖ, Monatschr. f. Ohrenh. **73**:615 (Sept.) 1939.

A tumor of the facial nerve, originating in the course of the nerve within the temporal bone, is a rarity. Such a condition is described, and as a matter of differential diagnosis it is pointed out that in such a case the paresis of the facial nerve is apparent for a long time when the drum membrane and hearing are still normal. The middle ear disease is secondary. Healing occurred in the reported case after radical mastoidectomy was done. Rejtö regards the tumor as benign.

LEDERER, Chicago.

SIGNIFICANCE OF THE COATED TONGUE. J. BERBERICH, Pract. oto-rhino-laryng. **2**:287 (Sept.) 1939.

The author discusses the clinical observation of the coated tongue both in normal persons and in diseased patients. He found that, normally, there is a marked variation in the amount of "furring" on the tongue. While furring occurs constantly in disease conditions, it is not diagnostic, in extent, degree or appearance, of any specific disease. The author has observed, however, that in association with all disturbances of the sensory fibers of the trigeminal nerve, irrespective of the cause—e. g., operation, injection of alcohol, suppurations of the petrous pyramid, intracranial tumor of the posterior fossa and herpes zoster—there is an unusual unilateral coating, which is generally ipsilateral. He also cites a case of a lesion of the precentral gyrus (the cortical center for the tongue) in which there was contralateral coating of the tongue.

PERSKY, Philadelphia.

EFFECT OF ROENTGEN AND RADIUM RADIATION ON THE ACTION OF CILIA WITHIN THE RESPIRATORY TRACT. PAUL FRENCKNER, Acta oto-laryng. **27**:297, 1939.

Frenckner investigated the effect of various doses of roentgen rays and radium on the ciliated epithelium of the upper respiratory tract. Ciliary movement was studied partly on excised pieces of nasal mucous membrane of guinea pigs and rabbits, partly on the mucous membrane of the maxillary sinus of living animals

and human beings in situ and partly on excised portions of adenoids. The speed and duration of ciliary activity were observed by measuring the movement of indicators laid here and there on the surface of the membrane and also by direct microscopic observation on the mucous membranes of experimental animals in vivo according to a new method and technic to be described later.

The experiments on animals as well as on human beings indicate that relatively small amounts of irradiation weaken or arrest ciliary movement. Ten observations were made on the excised mucous membrane of animals and on adenoid tissue, while 41 observations of the ciliary action of the nasal mucosa of human beings in situ were made. The effect of the same dose of radiation varied somewhat among different persons. In 15 human beings who received 50 mg. of radium for twenty minutes, ciliary movement was arrested for at least several hours. In 1 subject administration of 500 roentgens at a distance of 30 cm. resulted in cessation of movement for two and one-half months, while teleradium, 3 Gm. for ten minutes at 6 cm., suspended the activity of the cilia for one month.

GROVE, Milwaukee.

THE COURSE OF INSPIRED AIR AND THE POSSIBILITY OF ASPIRATION OF MUCOPUS FROM ONE SIDE OF THE NOSE INTO THE CORRESPONDING LUNG. H. A. E. VAN DISHOECK, Acta oto-laryng. **27**:414, 1939.

By causing coal dust to be inspired through one nostril and subsequently observing where it was deposited and also by producing inhalation of oxygen through one nostril and nitrogen through the other and analyzing the expired air chemically van Dishoeck came to the conclusion that the mixing of air inspired through both nostrils is perfect before it reaches the trachea, granted that both sides of the nose are of equal caliber. When, however, one side of the nose is narrow and the other is wide, the weak current of the narrow side will be carried along laterally in the strong current of the wide side and will not mix well. In such a case the current will sweep air from the narrow side of the nose to the lung of the same side and not into the other lung. He does not believe that the same thing takes place when mucopus is under consideration in cases of one-sided sinal infection, because the air current from the stenosed side of the nose is too weak to carry along the sticky mucopus. This can also be shown by having a patient with one-sided sinal infection forcibly exhale from that side of the nose through a tube having the same pattern as the nose, pharynx and trachea. If a glass slide is held at the end of the tube, it is noted that on normal and forced expiration droplets are not carried off with the air stream.

From these experiments Dishoeck concludes that pulmonary infection by means of aspirated drops in cases of nasal infection is of little consequence and that the chance of infection of the lung of the same side is slight. GROVE, Milwaukee.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland. Time: June 2-6, 1941.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
President-Elect: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Hotel Cleveland, Cleveland. Time: Oct. 6-11, 1940.

American Broncho-Esophagological Association

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.

American Laryngological Association

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Los Angeles. Time: June 1941.

SECTIONS:

Eastern.—Chairman: Dr. N. S. Weinberger, Robert Packer Hosp., Sayre, Pa.
Southern.—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn.
Middle.—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.
Western.—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Otological Society

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. Isidore Friesner, 36 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 26-27, 1941.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.